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SOME NEWER ASPECTS OF THE PATHOLOGY OF FAT AND FATTY DEGENERATION.[1]

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During the past few years numerous investigators have been engaged in the study of fat in tissues. New methods of investigation have been evolved, new facts discovered, errors in previous work detected, and gradually, as a result, our ideas as to the occurrence of fat in the body, its sources, and its significance have changed. From the laboratories of the chemist, the physiologist, the pathologist, and the experimental biologist contributions to our knowledge of the subject have come. These researches are of great interest, the methods used often very ingenious, and the results of much importance in the proper interpretation of pathological findings. Consequently it has seemed to me preferable to review this afternoon some of this more recent work and to discuss its interpretation rather than to limit myself strictly to the narrow field in which I myself have worked. The subject which I have selected is then a broad one and necessarily in the time at my disposal I must confine my remarks to its more important phases and even these can be discussed in but a superficial fragmentary fashion.

[1] A lecture delivered to the post-graduate classes of the Johns Hopkins University, May 9, 1904.

I. *Methods of technique.*—At the outset it may be well to speak of the methods and criteria at our disposal for the detection of fat in tissues. These may be divided into two groups: (a) those which visually demonstrate the fat within tissue, and (b) those which separate fat from tissue.

The latter group (b) comprises various chemical methods of analysis. They involve extraction of the fat by ether or other extractives in appropriate apparatus, an estimation of the quantities obtained, and a determination of the nature of the extracted substances.

In the first group (a) properly come those physical properties of fat and fatty tissue macroscopically evident, such as the tawny yellow color, greasy feel, indistinctness of normal markings, and increased friableness of the fatty liver; and those microscopically evident as the high refraction of the fat droplets and its solubility relations. These criteria, however, do not suffice; for small amounts of fat are not macroscopically evident; conditions of simple anæmia may be misinterpreted as fat; microscopically error may arise from the high refraction of substances not fat, while in unstained tissue little knowledge can be had of the relation of fat drop to cell struc-

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ture. Hence arises the necessity for staining methods, and these form the most important part of the first group.

Of staining methods there are two kinds, one giving a negative, the other a positive picture. In the first are all methods which preserve and stain the cytoplasm and nucleus while the fat is dissolved out, leaving a vacuole in its place. Such preparations give evidence of the relation of the fat droplet to cell structure, provided the fat droplet is larger than a certain minimal size, but they require other proof of the fatty nature of the substance from whose former presence the vacuole has resulted.

This proof is given in the use of certain stains which may be applied directly to the fresh tissue or to it after formaldehyde fixation. These stains are osmium tetraoxide (osmic acid), Sudan III, Scharlach R, and Indophenol.

Of these stains osmium tetraoxide has been longest in use and is the method still most generally used for the detection of fat. It is reduced, probably to metallic osmium, by the fat and the fat droplet appears of a dark brown to black color. It can be employed in a variety of well known combinations. The chief advantage of its use is the fact that fat after treatment with it becomes insoluble in the fluids necessary for embedding in celloidin and paraffin. Its disadvantages are its imperfect penetration into blocks of tissue, the fact that it stains other substances than fat, and does not stain all forms of fat. The latter is very important and was first emphasized in 1894 by Altmann who showed that osmium tetraoxide stained only olein and oleic acid and was to be regarded solely as a reagent for the demonstration of these components of fat. That osmium does not stain all forms of fat has been proven by a number of investigations since then, though there has been some discussion concerning the chemical nature of the fat to which its action is limited. As a single example of this limitation in the use of osmium tetraoxide, I found in the cells in the early stages of pneumonic exudations much fat present in small droplets which failed to stain by the osmium method either directly or after secondary reduction in alcohol but stained characteristically with Scharlach R and Sudan III. Further I showed that this was not due to the physical condition of fineness of emulsion, but to some peculiarity in the chemical composition of the fat in question.

For the above reasons the general use of osmium tetraoxide in the past has led undoubtedly to error in our estimates of fatty changes, and after its use alone conclusions as to the presence or absence of fat in a tissue should not be drawn. Yet used in conjunction with other methods it is of much value in the study of pathological processes.

Sudan III and Scharlach R, the former introduced into microscopic technique by Daddi (1896), the latter by Michaelis (1901), stain fat red. They can be used either on fresh tissue or after formaldehyde fixation. Tissue so stained can be mounted in glycerine. These compounds stain, so far as our present knowledge goes, all three forms of fat and only substances of a fatty nature. Of the two Scharlach R is preferable because it stains fat more intensely, tints the other tissues less, and is less likely to precipitate in tissue than Sudan III. It is best used in a saturated solution in 70% alcohol in which the sections are stained 24 hours to 48 hours or in a saturated alkaline alcoholic solution for a shorter time, a method recommended by Herxheimer. Of the two methods I personally prefer the former.

Sudan III and Scharlach R seem to be the best fat stains at our service. The disadvantage of them is the necessity of application to frozen sections and mounting in glycerine. These stains merely demonstrate the presence in tissue of certain fatty substances without indicating their chemical nature. The presence of these substances in tissue indicates certain metabolic activities of the cells to be discussed later.

Indophenol stains fat blue. It seems not to have been extensively used, and I have had no experience with it.

II. *Adipose tissue.*—In the body, fat is found in certain regions in the form of adipose tissue. In addition it is present in many of the organs and tissues under normal conditions. The chief sites of adipose tissue are in the subcutaneous tissues, in the omentum and mesentery, retroperitoneally, especially about the kidney, and to a less extent beneath the pericardium and parietal pleura. In these situations the fat is often present in very large amount and these sites form in the body the normal storehouses for fat and represent the true fat organs.

Here the fat cells are closely packed to form lobules of irregular shape and varying size. Between the lobules there is a moderate amount of connective tissue in which course the larger blood vessels and lymphatics. Between the individual cells the connective tissue framework is reduced to a minimum. With the anilin blue connective tissue stain only here and there fine fibrils appear, while with the phosphotungstic acid hæmatein stain a few scattered fibroglia fibrils can be made out, generally in association with connective tissue cells lying in close apposition to a capillary blood vessel. Elastic tissue occurs only in association with the larger blood vessels. It is thus evident that the cells of adipose tissue, of mesenchymal origin, have almost entirely lost their power of forming fibrillar intercellular substance. Instead they seem only concerned in fat metabolism and in this sense are specialized in function.

The most striking feature of adipose tissue is its great vascularity. This is especially evident where for any reason the fat is congested. Then between almost every cell can be seen a thin walled capillary—a surprising richness in vessels if our ideas have been based on the ordinary uninjected specimen of fat. This very intimate relation between blood capillary and fat cell must be very important in the metabolic functions played by the fat tissue. The adipose tissue is not, I think, to be regarded as merely a passive storehouse for fat but rather as a diffuse fat vascular organ in which great activity of fat synthesis or fat analysis may take place.

Fat cells vary somewhat in appearance though in the adipose tissue of the adult the common form is a large cell containing a single large fat droplet. The cytoplasm is reduced to a thin rim or membrane which takes a violet tint

in specimens stained by the anilin blue connective tissue method. This rim is almost homogeneous in appearance. The nucleus is crowded to one side and flattened out to accommodate itself to the curvature of the fat droplet. About it is a small amount of granular cytoplasm which often contains bright yellow granular pigment. Within the nucleus there is present almost always a single or several fat droplets, crowding aside the nucleoplasm and chromatin of an otherwise vesicular nucleus. These fat droplets are usually small (1 to 2 micra) but often are larger, may exceed in diameter the thickness of the nucleus and present as a distinct boss on one or both surfaces. In nuclei at times they are so numerous that the nuclear substance is present merely as a delicate reticulum enmeshing the fat droplets. Their very common occurrence in the nuclei of fat cells and the absence of any evidence of injurious action on the chromatin makes it very certain that they are not to be regarded as a degenerative phenomenon of the nuclei. Their presence often aids in distinguishing the nucleus of the fat cells from those of the scattered connective tissue corpuscles between them, for I never have found fat vacuoles in the nucleus of a cell distinctly between the fat cells and of connective tissue character or in the endothelial cells of blood vessels.

The cell described above is by far the most frequent in adipose tissue and in lipomata, but another type occurs. This is a cell slightly smaller, containing not a single fat droplet, but several, often many. In these there is proportionately more granular cytoplasm and the nucleus is not so crowded to the periphery though generally occupying the outer part of the cell. Sometimes the nucleus is in the center. Such cells occur singly, but much more often in groups of three or four to a dozen or more and were found more often in the fat of the omentum and the perinephric region than in subcutaneous adipose tissue. They resemble closely the fat cells occurring in the "brown fat" of the rat and other mammals, in the mesenteric fat of a fœtus at term which I have recently examined, and in a peculiar lipoma of the neck excised at the Boston City Hospital. Their exact significance cannot be stated, but as in some cases transitions to the first or common type can be made out, it seems probable that they are cells showing very active fat metabolism, and more probably fat synthesis than fat splitting.

Not infrequently in fat tissue or about small groups of fat cells occurs a third type, smaller than either of the two preceding types of cell and filled with very many small fat droplets. The nucleus of this cell is vesicular, centrally situated and has never been seen to be vacuolated. Such cells are numerous in adipose tissue undergoing repair, such as healing abdominal wounds. Here they occur singly or in groups about fat droplets. Sometimes they are larger and multinucleated. They also occur about the fat cells of the bone marrow in certain infectious diseases, as small-pox. Scattered groups of them I have seen in one specimen of lipoma and numerous cells of similar appearance were found in a specimen of breast in an inflammatory focus, associated with tissue destruction and the escape of colostrum. From the size, structure, and occurrence of these cells they seem to be of endothelial character and are probably concerned in the removal of fat set free in the tissues by injury of the fat cells —are phagocytic. This explanation seems more in accord with their occurrence than that they are young fat cells concerned in the regeneration of adipose tissue.

III. *Fat in organs and tissues under normal conditions.*—Before considering the fat normally present in the body other than in adipose tissue, it is necessary to emphasize the necessity of distinguishing between fat that can be visually demonstrated in a tissue and fat that can be extracted from that same tissue. A single example is sufficient to make this difference clear. In the normal human kidney almost no fat can be demonstrated in the cells by any of our staining methods; yet by the method of extraction the kidney is shown to contain under normal conditions a very considerable amount of fat, according to Rosenfeld, from 16 to 23%. In a diseased condition on the other hand, the kidney cells under the microscope may show a rich supply of fat droplets, an evident difference as shown by the microscope from the normal kidney. However, from that same kidney, chemical analysis may yield no more fat than from a normal kidney. There is then an evident difference between the results given in the two methods and the presence or absence of fat as determined by one method or the other, obviously has a different significance. Consequently the method used in a given case must be stated in order to avoid confusion in interpretation of results.

Fat capable of visual demonstration is known, however, to occur very much more generally in the body tissues than was formerly held. This knowledge has come mainly since the introduction of the stains, Sudan III and Scharlach R and is the result of work of Sata, Stangl, Fischer, Herxheimer, Hansemann, Cordes, Erdheim, and others. They have shown that fat is present in almost all of the glands of the body and occurs there under normal conditions in some way associated with cell metabolism.

The almost constant presence of fat in the liver has been long recognized and its large amount distinguishes it from other organs, so that the liver is very generally regarded as a fat depot analogous to the adipose tissue. Perhaps here the fat may undergo some change preparatory to its utilization by the other cells and so stand in a different relation to liver cells. That fat is normally present in the epithelium of the salivary glands, pancreas, adrenal, testis, ovary, thyroid, thymus, sweat and sebaceous glands, and breast is not so generally recognized. Of particular interest to the pathologist is the question of the occurrence of fat in the kidney and heart.

In the kidney of many of the lower animals fat occurs under normal conditions, particularly in the straight tubules where it has a basal position in the cell. In the human kidney it seems that small amounts of fat may be normally present and that slight amounts of fat cannot without other evidence be considered as pathological. However, any considerable increase of fat may be regarded as pathological.

Practically the same holds true of the heart muscle where Ostertag has found fat in normal guinea pigs and pigeons

and Fibiger in normal cats. Lubarsch has shown that fat is associated with the perinuclear pigment of the heart muscle as it is with the intracellular pigment of nerve cells. Consequently in these two most important organs, the heart and the kidney, it is not the finding of small amounts of fat which can be interpreted as indicative of pathological change, but the occurrence of more than a certain minimal amount of visually demonstrable fat, at the same time remembering that fat can be chemically extracted from both organs under normal conditions in considerable amount. Of the other tissues, skeletal muscles, endothelium of blood vessels, connective tissue corpuscles, and leucocytes generally contain fat. Herxheimer, reviewing the question, finds of all the tissues, the spleen and lymph nodes are almost the sole ones that are normally fat free.

In most organs where fat occurs it is very scant in fœtal life if present at all but gradually increases in amount after birth. Such fat can be regarded in no other light than the result of the metabolic activity of the cells and bears no relation to cell degeneration.

IV. *Fat in tissues under abnormal conditions.*—Of far greater interest is the occurrence of fat in tissues under abnormal conditions. Many studies have been made of this subject and the meaning of the findings has been the subject of much discussion. The frequency with which considerable amounts of stainable fat is found in the liver, heart, and kidney at autopsy is generally known, but other organs have not been so systematically studied. For several years at the Boston City Hospital we have made systematic examinations of the heart, liver, and kidney, at first by means of teased specimens or frozen sections treated with acetic acid, during the past year or so by means of the Sudan III and Scharlach R technique. This has impressed us with the great frequency of the finding of fat in autopsy tissues.

In our last 50 autopsies the heart, liver, kidney, and spleen have been studied by the Scharlach R method. These 50 cases may be roughly classified as follows: In 25 the immediate cause of death was some acute disease such as pneumonia, peritonitis, or diphtheria; 17 showed chronic processes; 8 were chronic processes with a definite superimposed acute infection. Only four bodies were emaciated, and even here there was considerable remaining subcutaneous fat. The hearts of these cases showed fat within the muscle fibres of the left ventricle in 56 per cent of the cases; in 28 per cent in slight amount, in 20 per cent in considerable amount, and in 10 per cent in very large amount. Of the kidneys 74 per cent showed fat, 38 per cent in slight amount, 30 per cent in moderate amount, and 6 per cent in large amount. Of livers only two were fat free, while 40 per cent showed a slight amount, 50 per cent a considerable amount, and 4 per cent a very large amount of fat. In 39 of these autopsies the spleen was studied; 18 were fat free, 21 contained small amounts of fat. In none of these cases were all of the viscera fat free, but in several fat was present only in the liver.

In the heart the fat was found in most cases not throughout the tissue, but in groups of fibres. Likewise in the kidney the localization was almost always a focal one and where the fat was very slight in amount it occurred most frequently in the basal end of the epithelial cells of the loops of Henle or of the collecting tubules. In the spleen the fat was found as a rule either in the capsule and trabeculæ, or in the endothelium of the blood vessels. Only in five cases was it elsewhere, once in the pulp cells, and four times in proliferated endothelial cells in the centers of Malpighian bodies. These last four were cases of diphtheria.

In the heart, kidney, and spleen the fat was practically always in the form of fine droplets, in the liver it was commonly in large droplets or as a mixture of small and large. In only eight cases were fine droplets alone present in the liver. Two of these were chronic, the other six acute cases, pneumonia or diphtheria.

The fat then has been associated with a very great variety of processes now chronic, now acute. What has been most striking is the great variety in distribution in the various organs in a given case and in different parts of the same organ or tissue.

In 28 consecutive autopsies I have studied the fat in the myocardium, pectoralis major, and psoas major muscles. Portions were taken from the papillary muscle and intraventricular septum of the heart and from two parts of the above mentioned skeletal muscles. These examinations showed that not infrequently the myocardium exhibited marked fatty change while the skeletal muscles were little affected or the converse. Sometimes there was a considerable difference between the two skeletal muscles. The figures were:

	Fat absent.	Fat present slight amount.	Fat present moderate amount.	Fat present large amount.
Heart	5	10	4	9
Pectoralis major	5	9	6	8
Psoas major	8	6	7	7

As a result of these and other studies there has been a growing feeling of a very imperfect knowledge of the causes underlying the distribution of the fat in a given case and the meaning of its presence. That this feeling is a very general one is evidenced by the present active discussion as to the sources and significance of intracellular fat.

Under the influence of a variety of conditions, such as general or local anæmia, venous stasis, phosphorus, arsenic, and lead poisoning, acute infectious diseases, many bacterial infections, fat appears in cells in increased amount. To the fat various origins could be assigned. This fat might be formed within the cell either from proteid or from other constituent of the cell. It might come from without the cell being transported thither by the lymph or blood stream.

In 1847 Virchow taught that the fat of fatty degeneration was formed within the cell as a result of the destruction of cell proteid. This dictum was accepted by pathologists until very recently and is yet the explanation very generally given in text books on pathology. What evidence is there for such an assumption?

Voit and Pettenkofer as a result of their work in 1862-71 claimed that the body fat was formed from ingested pro-

teid. Voit had fed dogs on lean meat and in excreta regained all the nitrogen of fed muscle proteid, but not all of the carbon. Of the carbon a greater part remained in the body, more than could have been converted into glycogen, and so he assumed that it was stored as fat. This was accepted until Pflüger in 1891 vigorously attacked these views. He showed that Voit and his pupils had based their calculations on an erroneous assumption of the carbon and nitrogen content of the lean meat, and that by using the correct figures the retained carbon disappeared, and with it the bases of Voit's assumption of the formation of body fat from food proteids. Since then numerous metabolism investigations have been made, the results of most of which negative the assumption of Voit, so at the present day it seems proven that body fat is not formed from the ingested proteid, but from food carbohydrates and food fat. Voit had further claimed in support of his view a formation of fat from proteid in adipocere, the ripening of cheese, and the secretion of milk. Later work has shown the first two to be due to bacterial activity, while the fat of milk is known to come from the body fat. So in physiology there is no evidence of a formation of fat from proteid in the sense of Virchow, other than the result of vegetable life, a condition which evidently does not enter into the present consideration.

One by one the various pathological conditions regarded as typical examples of fatty degeneration have been submitted to the most careful microscopic and chemical scrutiny or subjected to the proof of animal experimentation.

In cases of phosphorus poisoning the cells of the liver and other viscera become filled with fat droplets. It was long held that this fat was formed from the proteid of the liver cell. In 1883 Lebedeff combated this and claimed that the fat came from the fat depots and did not arise within the cell itself. He based his claims on finding linseed oil after phosphorus poisoning in the liver of fasting dogs which he had previously fed on linseed oil. Rosenfeld by a series of careful experiments, the first reported in 1897, demonstrated the transportation of fat in phosphorus poisoning. He starved dogs until their body fat was reduced to a minimum. He then fed them on lean meat and mutton fat until their fat depots were filled with mutton fat. Again these dogs were starved until no fat was in the liver, that disappearing very much earlier than the fat of the fat depots. The administration of phosphorus now brought about a marked appearance of fat in the liver. Analysis showed this fat in the liver to have the composition of mutton fat which points to a transportation of fat from the fat depots, not a formation from the proteid of the liver cells, in which case the fat would have had the composition of that normal for the dog. Further in starved dogs, whose body fats have been used up, phosphorus poisoning produced no fatty degeneration because there was no fat for transport. Fibiger obtained the same results in chickens after phosphorus poisoning and in rabbits after diphtheria toxin. As early as 1864 Leisering had noted his failure to get fat in the liver after giving phosphorus to two lean chickens. Clinically this is supported by the absence of fatty liver after phosphorus poisoning in a very lean patient reported by Lebedeff from Kussmaul's clinic. That the fat comes from the body fat by the way of the circulation is supported by finding the fat increased in the blood in cases of phosphorus poisoning, observations of Méhu, Rosenfeld, and Daddi. Various objections raised to the experiments of Rosenfeld have been one after another successfully met, and his results confirmed by others. Analysis of these livers shows, besides changes in the fat, a disappearance of glycogen (Saikowski, Luchsinger, Stolnikow, and others) and the formation of split products of proteids. The latter indicate cell degeneration, but too little to account for the fat—the two processes fatty infiltration and cell degeneration go hand in hand. Very similar results have been obtained after the action of other poisons as arsenic, chloroform, and phloridzin. Leick and Winkler, using this method of Rosenfeld, have shown that the fat in the heart after phosphorus poisoning originates from body fat by transport.

Athanasiu and Taylor attacked the problem in another way. They used frogs. Taylor in his experiments took a dozen frogs and divided them into two groups, each of which had the same number of frogs and the same total weight. To one group he gave phosphorus, the other remained as controls. When a phosphorus frog died a control frog of equal size was killed. The bodies of all were dried, pulverized, and their fat extracted. The phosphorus frogs had fatty livers but their total fat was not increased which is evidence of a redistribution of fat—not a new formation.

There is also much morphological evidence of fat transportation. Fat is often seen deposited in that part of the cell adjacent to a blood capillary. This has been noted in the adrenal, is not infrequent in the liver, while in the kidney the basal end of the cell is the common locus of the fat. In renal infarcts it is the boundary zone, where circulation or fluid transfusion is maintained about injured cells, that accumulates fat, while the center of the infarct and the uninjured parts of the kidney remain fat free. Fischler in his studies of this condition concludes that the appearance of the fat depends on the circulation; there is no fat deposited where there is no circulation, and further, it depends on the cells themselves; injured cells take up fat, but they must be living, not dead cells. Fat comes in a soluble form and the cells must be possessed of metabolic activity in order to synthesize this material into demonstrable fat droplets. The same relation of fat deposition holds for tuberculous caseous areas, about which there is a boundary zone of fat. Hagemeister in a study of granulation tissue, tuberculosis, kidney infarcts, etc., places more stress on the relations between fat appearance and circulatory disturbances, and even minimizes the part played by the cells themselves. He thinks fat disappears where there is an increased flow of blood and lymph, but is taken up by cells where there is an obstructed flow. Arnold as a result of numerous experiments in which he injected fats, soaps, etc., emphasizes strongly the transport of fat in so-called fatty degeneration.

Fischler in experiments on isolated kidneys showed the synthesis and deposition of fat in the tubular epithelium from soaps circulated through the organ. In my studies of pneumonia I found the same fat within the polynuclear cells of the circulating blood which I found within the cells of the early exudates and believed that the leucocytes arrived in the exudate already containing the fat.

As a result of all of these observations, it seems that, in a variety of conditions, formerly regarded as true fatty degeneration (that is, a formation of fat from proteid), the fat arises from without the cell, is transported thither. Does all of the fat so arise? This is very much more difficult to prove, but seems to be answered for the liver under certain conditions by the following work of Rosenfeld. Dogs with fat depots stored with mutton fat were prepared as above and then starved to remove the fat from their livers. A part of the liver was then excised, the amount of fat determined and its iodin number found. The animals now received phloridzin or ol. pulegii, after which their liver fat and body fat were similarly analyzed. The results are shown in the following table:

	Leber.					
	Exstirpiertes Stück.		Uebrige Leber			Depotfett
	Fett %	Jodzahl	Fett %	Jodzahl gefunden	Jodzahl berechnet	Jodzahl
A. Phloridzin ...	16.7	106.5	30.0	78.8	81.4	49–52
B. Ol. Pulegii...	14.03	71.3	19.8	61.2	66.2	53.8
C. Ol. Pulegii...	22.5	67.8	33.33	60.8	62.2	50.4

An examination of this shows a close agreement between the calculated iodin number of the liver fat and that actually found after treatment—a very satisfactory proof of the origin of all the fat of the liver from the fat depots under these conditions.

Against the assumption, that all fat is transported to the cell, are the careful analyses of abnormal kidneys in which Rosenfeld found no considerable increase of extractive fat though microscopically there was the condition of fatty degeneration. Similar results have been found for areas of cerebral softening. In tissue preserved aseptically without the body according to many observers fat droplets appear (autolysis). Here there is no circulation. Consequently it seems that we must assume a formation of fat within the cells. There is no evidence of a formation of fat from proteid though the possibility cannot be denied. Fatty bodies do occur in organs where we cannot detect them microscopically. They may be in a state of such extremely fine division as to escape detection—a change of physical condition would give their appearance as fine droplets. They may be in a loose combination with proteid or other substance—a combined fat not visually demonstrable which under certain conditions splits into free fat and some other body. Taylor has furnished some evidence of this. We may have compounds such as protagon or lecithin in whose complicated molecules fatty radicals exist to be later split off. In some such form it seems fat-like or fat-related bodies exist in protoplasm and at present we are not justified in deriving all fat that appears pathologically from sources without the cell.

Of fatty degeneration in the sense of Virchow we have no proof. At present our knowledge is in too unsettled a condition to be justified in the creation of new terms. We must wait until chemical and experimental study can give us more data before we can create new names which will perfectly harmonize with the facts of the case. For the present it seems best to continue the use of the old terms—fatty infiltration and fatty degeneration, and define the latter as the appearance of fat in cells which have undergone some form of injury. This fat arises from without and is transported to the cell or arises within the cell from fat-related substances. The former probably explains the greater part of the fat, though in this respect tissues may vary. At present there is most evidence for a purely extracellular origin of fat in the liver. very much less for the kidney. Conditions determining the one and the other need further study.

Here I must leave the subject. I realize how imperfectly the ground has been covered, but I trust the following points have been sufficiently emphasized:

(1) That osmic acid does not stain all forms of fat and fat alone, and so is but an imperfect method of demonstrating fat. Sudan III and Scharlach R, though having disadvantages, give more satisfactory results. (2) That visually demonstrable fat is present normally in very many cells of the body while extractive fat occurs in practically all of the tissues. (3) That under abnormal conditions visually demonstrable fat appears in cells in increased amount and is an index of cell injury. (4) That fatty infiltration is the physiological appearance of fat in normal cells and fatty degeneration is the appearance of fat in injured cells; the fat is an index rather than the direct result of the cell degeneration. (5) That in both, the origin of the fat is probably the same and mainly from without the cell by transport from fat depots elsewhere, but may arise within the cell from fat-related bodies, not from proteid.

STUDIES IN TYPHOID FEVER.

SERIES I-II-III.

The papers on Typhoid Fever, edited by Professor William Osler, M. D., and printed in Volumes IV, V and VIII of the Johns Hopkins Hospital Reports, have been brought together and bound in cloth.

The volume includes thirty-five papers by Doctors Osler, Thayer, Hewetson, Blumer, Flexner, Reed, Parsons, Finney, Cushing, Lyon, Mitchell, Hamburger, Dobbin, Camac, Gwyn, merson and Young. It contains 776 pages, large octavo, with illustrations.

The price is $5.00 per copy. Address The Johns Hopkins Press, Baltimore, Maryland.

FABRICIUS GUILHELMUS HILDANUS: THE FATHER OF GERMAN SURGERY.[1]

BY WALTER B. PLATT, M. D.

The father of German surgery, or as he has been called the Ambroise Paré of Germany, was born on June 25, 1560 in Hilden, Germany, not far from Düsseldorf, as plain Wilhelm Fabry. Early in his professional career he latinized his name after the fashion of the day, making it Fabricius Guilhelmus Hildanus, now often called simply Hildanus, to distinguished him from another Fabricius. As Latin was the international tongue of letters, religion, and what there was then of science in Europe, this was not as absurd and pedantic as it now seems. The fraternity of letters was far less national than at present, and students and scholars went from land to land seeking the knowledge they could not otherwise obtain. For these reasons most of the important works of Hildanus were written in Latin.

His father was clerk of one of the courts, a man of parts we hear, and at least respectable and respected. Hildanus himself speaks of his father as "homo literatus."

The father dying when Hildanus was but a year old he was left to the care of his mother who speedily remarried. Of our surgeon's boyhood we know little except that he seemed to have unusual vitality, vastly enjoyed swimming, and suffered a paralysis of the tongue at the age of twelve in consequence of swimming too long in cold water. It was many years before he perfectly recovered his speech. Left with little or no means early in life the good poet Utenhof assisted him, sent him to school in Cologne, and as he grew older urged him to become a physician. Medicine was not at all to his taste and from the first he preferred surgery. In 1576 when but 15 years of age he entered the service of a surgeon named Dumgens at Neuss, with whom he remained four years, after which he joined himself to the surgeon of the Duke of Cleve. Now began a series of wanderings from place to place.

Going to Metz in 1585 he went thence to Geneva where he operated on a strangulated hernia. Two years later he married Marie Colinet, who turned out to be a helpmeet indeed. Leaving Switzerland in 1588 he returned to Hilden for 3 years, going thence to Cologne where he practiced from 1591 to 1596.

Besides practicing surgery arduously, he cultivated the science of medicine in its broadest sense, and wrote many monographs and descriptions of surgical procedures. In 1593 he published what is perhaps the best known of his writings at the present time, "De Gangrena et Sphacelo." He also described the most important of the valvular folds of mucous membrane of the rectum, and demonstrated its function.

We find him again at Lausanne in Switzerland in 1596 where he worked hard at his anatomy and made many autopsies, but he could not remain very long in one place and went back to Cologne in 1597 where he remained 3 years. He was in Lausanne again in 1600, whence he fared to Payern. In 1607 he published a work on dysentery, and constructed a model of an eye for the use of students.

His family had the bubonic plague in 1613 and most of his children died of it. After he thought he was at last rid of it, the plague reappeared in his house after an intermission of months.

About this time von Hildanus became a great sufferer with a neuralgia or gouty headache, which he finally traced to a particular kind of wine of which he was very fond. He also was a martyr to gout.

Those who are afflicted in a similar way may console themselves with the thought that they are in good company for numbers of our greatest (as well as our laziest) men have had the gout. Hildanus now lived in Berne for a season. In 1619 he finished "Observationum et Curationum Chirurgicarum Centuriæ IV," and wrote his hundred letters to his friends.

He wrote "Observationum et Curationum Chirurgicarum Centuriæ VI," his last, in 1623, and in 1620 on a "Christian way to Eat." In 1624 he wrote upon the value of anatomy.

In 1626 he wrote his well-known work on Stone, and two years later was very ill with the gout.

After this it is quite natural, that he should write upon the "Preservation of the Health" by the use of certain warm springs.

We think we can see the steady progress of gouty nephritis as in 1631 he had bronchitis, and was often annoyed by asthma.

He died on February 14, 1634, aet. 74 years.

Of all his children, but one, a son, survived his father.

Hildanus was a deeply religious man, a very conscientious operator, not at all vain; on the contrary he was always dissatisfied with his work, while the amount he accomplished was prodigious. He always declined to operate when he thought the outlook a fatal one. His letters were very numerous. He is said to have corresponded with 150 physicians and other learned men. Although very modest he did well financially, unlike most men of his type. He always showed great mental activity.

HILDANUS.

In the time of Hildanus, Wesling writes from Venice comparing German surgery with that of Venice, saying "Surgery has a somewhat better status here, although even here there are those called surgeons who come from the dregs of the population, so that if Venice had to take the field, about 2000 dirty barbers and enema givers would enlist as surgeons."

[1] Read before the Johns Hopkins Hospital Historical Club, March 14, 1904.

Hildanus gives a few examples of the state of surgery in his time as follows:

"A cabinet maker who was watching the drilling of an artillery battery, was wounded in the breast by the discharge of a blank charge from standing too near, in consequence of which the covering of the chest was torn away down to the ribs. A barber surgeon who was summoned, seeing a bare rib, seized it with a pair of forceps, and tried with all his force to drag it away, thinking it some part of the ramrod. Fortunately a bystander drove the barber away, applied a suitable dressing to the exposed ribs, and in time the man recovered."

Hildanus says "In spite of all their ignorance, the surgeons thought they knew better than educated physicians" and he relates how in one case where he "had refused to amputate the limb of a man who had senile gangrene, a barber did the operation, amputating the thigh which of course speedily became gangrenous at the seat of operation, whereupon the patient died;" Hildanus writes further: "Christian pity often makes me weep over the doings of those men who play so recklessly with human life." (Archiv f. klin. Chirurgie, 1865, vi, by Meyer Ahrens.)

Although a surgeon, Hildanus knew his anatomy better than most professors of that branch, and laid great stress upon a thorough knowledge of this subject. He called anatomy the "key" to every department of medicine, and the "rudder" to all medical practice.

He demanded that priests should have some knowledge of it, and that lawyers should likewise have an acquaintance with anatomy that they might apply torture with more effect and discretion to criminals, so as to avoid sudden death, or a confession of crimes which had not been committed. He saw the importance of some education of midwives in anatomy that they might know enough to call in a physician before it was too late to do any good.

At the end of the 16th and beginning of the 17th century it was difficult to obtain bodies to dissect in Switzerland. In the year 1601 when he demonstrated the valvulæ Bauhini (of the rectum) on a cadaver, it is related that he carefully preserved the fat of the subject to send to a French physician who wished to use it as a therapeutic agent.

Hildanus' first good anatomical preparation was a maceration, showing the blood-vessels of the abdominal viscera, notably those of the liver, kidneys and genito-urinary system.

According to the custom of his time, he finds all manner of curious analogies between the human body, and the universe in general.

He makes gruesome remarks upon the advisability of so applying torture to criminals as not to fracture the scapulæ by bending back the arms too far, something he has often seen, since men thus treated would be apt to confess to crimes they had never committed.

Like a true scientific surgeon he attached great importance to a knowledge of pathological anatomy. He took an especial interest in congenital malformations, making a number of drawings in these weird creations. He made moreover a collection of bones showing the healing of fractures, and in a letter to Prof. Paw he eulogized the value of Comparative Anatomy, Botany and Chemistry.

Like most physicians of his time he was a believer in the medicinal virtues of various disgusting agents, such as portions of vipers, and of dried toads. Mineral water baths were objects of his admiration as healing agents, and he wrote a monograph in 1629 on the preservation of health by the use of certain thermal springs. He wrote much about the plague or what he thought was the plague, which destroyed so many human lives and spoke of the inefficacy of any known treatment, although he felt sure that those who had issues in their arms did not die of it.

Hildanus was a great believer in the use of minute varieties of instruments, and spent much time and ingenuity in devising them. One of these was a bullet extractor, consisting of a tube which was introduced down the track of the bullet until it reached the ball when a cork-screw-like device was protruded. This was supposed to enter the ball, so as to allow of its removal by traction. He treated the bites of rabid animals with all sorts of irritating mixtures, but also with the actual cautery.

He combated the idea advanced at that time, that in cranial fracture the depressed bone should not be elevated, and invented a somewhat impracticable instrument to raise the depressed fragments. He makes frequent use of egg albumen as a styptic, discusses polypi, and describes the operation for the removal of the breast for cancer. As far as the anatomical part of it is concerned he operated much as we did 20 years ago. The use of caustics in these cases which had a charm for many patients and physicians, he deplored.

Concerning the radical cure of inguinal hernia, he remarks the extreme rarity of such cures in his time, but details one in a well-known personage where such a result was achieved by several months rest in bed in a horizontal position and the continual use of a truss.

It is interesting to note that Hildanus was a firm believer in the virtues of issues, and fontanelles, devices to secure suppuration, as a means of warding off disease by providing an outlet for "vicious humours" which were supposed to be their cause.

He invented a suspensory bandage, and urinals to be worn by the patient when going about, and also club-foot apparatus. Fabricius was aided by a help-meet indeed, who was skillful in the art of obstetrics, for his wife was often present in difficult cases.

At the beginning of the 17th century no one thought of calling in a physician until midwives had utterly failed. It was then usually too late to do much for the patient, for these midwives were a wretchedly ignorant lot. In a Treatise on Lithotomy published in Basel 1629, Hildanus warns all ignorant persons not to undertake to cut for stone, and in the course of his discussion of the operation he says, "The patient should prepare himself in all ways for death before the operation, he should make his will and ask for prayers in church." That Hildanus had an exalted ideal of a surgeon will be seen when we learn that "he should before all, fear God, be

true, pious, upright, not given to cursing or swearing, he should not be addicted to drinking, whores or gambling." "The patient should avoid all physicians who gossip or chatter much, or who boast of their powers as if none were equal to them."

Furthermore he says "a surgeon should not be merely an empiric guided alone by his experience, but should have studied more in the science and groundwork of his profession."

Hildanus operated for stone in the male bladder usually by a lateral incision in the perineum, but they were sometimes removed through the rectum, while in women he removed them either by way of the vagina or urethra. When patients came to the surgeon with very large stones, Fabricius advised that operation be declined, and that they be turned over to their spiritual advisers.

Stones in the bladder at this time were often removed in several sittings, the incision being the first sitting, when unless the stone presented at the opening, delay was advised until it did, when an attempt was made at extraction. If this did not succeed, another wait was advised, the patient being put through a course of medical treatment tending to relax the opening and eject the stone.

Meyer Ahrens mentions a "stone-cutter" of Ulm who operated between 1610 and 1626 upon 2000 cases of stone in the bladder and hernia, who was absolutely opposed to operating for stone in several sittings.

Perhaps the most famous work of Hildanus was one upon Gangrene, Hot and Cold, published in 1593, in which he advises that operation must be done by all means in sound tissue and if the gangrene be in the foot the amputation should be 4 to 5 fingers' breadth below the knee. If the gangrene be above the knee, as near as possible to the gangrenous part. He dwells much on the preparation for the operation by purgatives, by supporting the heart's action and sometimes by venesection. He advises that arteries be tied only in strong and full-blooded individuals. At that time, Harvey's discovery of the circulation of the blood had not been made, and the tourniquet was not used. Pressure forceps, which had narrow beaks, were used to compress arteries. When arteries were ligated we have the authority of Meyer Ahrens for the statement that this was done by applying ligatures over artery forceps. After the amputation, compression was applied to the parts.

In an essay "De Combustionibus" he makes 3 degrees of burns, and in treating of gunshot wounds he discusses the problem, whether or not we ought to take into consideration the heating of the bullet by its passage out of the gun, or in passing through the air, or whether a poisoning of the wound takes place from the gunpowder used.

A Latin edition of his works was published in Frankfort, 1646.

WORK OF FABRICIUS HILDANUS.

His works are mostly in Latin, some in French and a few in German.

His best known work that on Gangrene is in old French and was written in 1597. Accurate language was not then the fashion, and more stress was laid on regimen then, than now. His work on stone in the bladder has this title: "Lithotomia Vesicæ, that is, an accurate description of the stone in the bladder, showing the causes and pathological signs thereof, and chiefly of the method whereby it is to be artificially taken out, both of men and of women, by rectum. Written first in High Dutch by Guilhelmus Fabricius Hildanus; first translated into Latin by his Scholler and Communer, and now done into English by N. C. for the general good of the nation and particular use of the Society of Barber Surgeons, London, 1640."

"To the worshippful Companie of the Barber Chirurgians, John Norton wisheth encrease in skill, fortunateness in practice, and all other happinesse. How dangerous in practice the Curation of the Stone by section is, it is evidently evinced not only by reason, if we consider the structure and temperature of the part affected, but by experience also; seeing so few attempt it, and so many doe die who submit themselves to this manner of curation, although the operator perform his part; wherefore too great caution cannot be had in going about this operation."

Chapter 1st of this work by Hildanus begins thus: "The divine Hippocrates, Father of Physicians, hath not without cause in his book 'De Arte,' left us this aphorisme: 'The Physician which can discover the disease, may be able also to cure it, advising thereby all prodent sonnes of art that they should use all diligence and care to get an exact and accurate knowledge of the disease before they intend or think to prescribe an order for the cure.'"

He then advises surgeons to read up, "first the ancients, Hippocrates, Galen, Paulus, Aegineta, but the later writers also, Vesalius, Fallopius, Aquapendente, Ambroise Paré and others, to find out the theory and intention," for he says: "This advise I doe not causelessly set downe in the enhance of this translate, For I know there are some giddy headed and unadvised chirurgians which upon each slight signe and uncertaine suspitian of the stone commonly apply the incision knife to the bladder, boring and searching there, yet in the end, find no stone at all." Again to define a stone he says: "I say the stone is a preternatural, grosse, slymie, coagulated humor, brought into a stone of a thick matter, by a preternaturell heate and hidden quality of the bladder."

CHAPTER 2.

"Because every faithful Chyrurgion and Physitian is, bound before God the Chief Physitian, not only to cure the sick, but by his good councel to keep those that are cured in their health; not only to take away the stone, but to provide against the breeding of any new ones; therefore it is required principally that the Chyrurg know and be taught how and from what causes the stone in the Body of Man and especially in the Bladder, is generated and coagulated together."

He thinks that those who cut men for stone and find none, should be severely punished "and to be paid in their own

coyne, for it is written 'an eye for an eye and a tooth for a tooth;'" also "Besides all this it is chiefly required that the Lithotomist and Cutter of Ruptures should be well versed in the Anatomical part of physick."

We see that Hildanus was an advocate of the higher education of physicians and of their divine right to practice, by the following: "In which words the scripture doth distinguish who are lawfully called to this work and approved by the learned from those who not after the wonted manner enter in at the gate, but like thieves creep in at the windowes and back doores, and by whom the sick men are deprived of their health with their goods and estate.

Such are idle and unskilled Mountebanks, Juglers, vagabonds, executioners, and other detestable villanes of this sorte, whom many would scarce trust for a farthing, whereas in the meantime they trust them with their health, both of their body and soule; So farre the Divell hath blinded man."

"Who so ever therefore should be ungratefull to a faithfull and industrious Physitian, being ordained by God, whether in word or deed, much more by keeping back his reward (of which sort I have met with many) he may easily perswade himself that God will be avenged of him for the labour and trouble which the godly, honest and faithfull Physitian hath undergone for the sick mans sake."

Just before operation "the help and assistance of Almighty God is first to be seriously and devoutly implored by a lawful Minister (if any be present) or in his absence by some of the bystanders who if he please may use one of the prayers prefixed before my treatise De Gangrena et Sphacelo."

The patient is then to jump, or if an infant it is to be shaken, "that the stone may fall down toward the neck of the bladder."

Hildanus advises a perineal lateral operation, and not to attempt to remove a large stone, but to abstain from operation where the patient has no chance of surviving, "for this is better and more commendable in a faithful and diligent Chyrurg than (as some usually doe) to draw the languishing Patients as it were to the shambles."

He relates one of his surgical experiences as follows: "It so befell me at Paterniam when I was about to cut off the thigh of a man of forty yeares of age, and ready to use the saw and cauteries. For the sick man no sooner began to roare out, but all ranne away except onely my eldest sonne, who was then but little and to whom I had committed the holding of the thigh for forme onely; and but that my wife, then great with child, came running out of the next chamber and clapt hold of the Patients Thorax, both she and myself had been in extreme danger."

He then discusses the folly of employing diuretics before the operation.

If we summarize the character and work of Hildanus we see in him a very talented yet modest man of strong vitality and convictions, exceedingly industrious, truly religious, devoted to his profession, restless and migratory in his instincts; a man of great personal magnetism, who by his numerous intelligent observations and skillful operations, more than all his predecessors is entitled to be called the Father of German Surgery.

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THE IMPORTANCE OF TESTING THE OCULAR MUSCLE BALANCE FOR NEAR, AS WELL AS FOR DISTANT VISION.

BY SAMUEL THEOBALD, M. D.,

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With increasing experience in dealing with asthenopic eyes, I am more and more impressed with the importance of making in every such case a careful test of the muscle balance for near, as well as for distant vision.

Although in recent years so much attention has been paid to anomalies of the ocular muscles, the significance of the muscle balance in near vision does not seem to have received the recognition which its importance demands. The fact that there is often marked discordance between the behavior of the muscles in distant vision and in near vision appears not to have impressed itself upon systematic writers upon diseases of the eye; and, indeed, it is evident that not all of them are informed as to what is the normal behavior of the muscles when tested for the reading distance.

What constitutes orthophoria in distant vision everyone knows; but, in spite of the fact that it was long ago pointed

out, everyone, it would seem, does not know that, as determined by the vertical diplopia test, the normal state at 12 or 13 inches is an exophoria of 2° or 3° or even of 4°, and that a muscle balance which in distant vision indicates orthophoria is indicative really of a heterophoric condition when found at the reading distance.

In my papers upon "Subnormal Accommodative Power."[1] I have emphasized this point, and have also set forth what, as I view it, is the peculiar significance of esophoria which is present in near vision only, or which is more pronounced in near than in distant vision.

The results which I have obtained in this condition, to which I gave the name subnormal accommodative power, by following the rules laid down in my papers—prescribing at times for young asthenopic emmetropes convex glasses of considerable strength for near work, and for young hypermetropes stronger glasses for near than for distant vision or combining with their near lenses esophoric prisms—have been so eminently satisfactory that this practice is now as much a matter of course with me as the correction of astigmatism or of hypermetropia itself.

The opposite condition, in which there is present a considerable amount of actual exophoria at the reading distance, with normal muscle balance in far vision, a condition by no means rare, is of scarcely less importance, and calls as emphatically for glasses especially adapted for near work, that is to say for lenses which, in addition to correcting such refractive error as may be present, take into account the muscular fault.

Occasionally, in dealing with this condition of discordance between the far and near muscle balance, and having in mind the convenience of the patient, I prescribe bifocal lenses; and, when they see one of my, perhaps, fifteen- or sixteen-year old patients thus equipped, some of my brother oculists are doubtless not a little surprised.

It is not my present purpose, however, to dwell upon these differences of lateral muscle balance to which I have alluded. It was the observation recently of several cases of hyperphoria, which was present in near vision only, and which proved to be an important factor in the causation of the asthenopic symptoms, that prompted me to write this paper.

Thanks to Maddox, we are able now to detect the existence of hyperphoria, and to determine its degree, much more easily and exactly than was formerly possible. With the aid of his multiple rod, and with the further assistance of Dr. Schild's well-contrived little electric light,[2] which I have the pleasure of showing you, I now find it no hardship to test for this defect in near as well as in distant vision. And the outcome of this is, I have learned that differences exist between the vertical muscle balance in far and in near vision much oftener than I had supposed. Indeed, I have not only found cases such as I have mentioned, in which hyperphoria was present in near vision only, but I have met with a few instances, not, however, marked in degree, in which there was hyperphoria in one direction in distant vision and in the opposite direction in near vision—a right hyperphoria, for example, at 20 feet and a left hyperphoria at the reading distance.

I am, therefore, as I said at the outset, more than ever convinced of the importance, in every case of asthenopia, of testing the muscle balance for near vision. Frequently this test shows nothing that is significant; but, on the other hand, it occasionally reveals a fault which must be taken into account if our patient is to obtain complete relief.

I may add, in conclusion, that in determining the balance of the muscles in near vision I use the simplest contrivances—for the vertical diplopia test, a prism of 7° taken from my trial case, and upon a card attached to a rod 12 inches in length a small object calculated to stimulate accommodation, such as an asterisk; and for discovering hyperphoric faults, a multiple Maddox rod with a Schild electric light. The Schild light, if one wishes, may be used instead of the asterisk in the vertical diplopia test; but I do not feel quite sure that it prompts the patient to accommodate as accurately as the asterisk does.

The employment of these tests, which usually give entirely trustworthy results, requires but a very few moments, and the information which they afford is, I am sure, much too valuable to be ignored.

[1] Trans. Am. Ophthalmolog. Soc., 1891 and 1894.

[2] Described in the Ophthalmic Record, June, 1904.

INFANTILE PARALYSIS OF THE ABDOMINAL MUSCLES; WITH REPORT OF A CASE.[1]

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The commoner distribution of the residual muscular palsies in acute anterior poliomyelitis of infants is too well-known to justify repetition. Nearly every monograph or text-book on the subject gives tables showing the relative frequency of the muscular involvement, but very few consider the question of the abdominal muscles. In looking over the literature, so few cases have been found as to justify the conclusion that a residual paralysis of the abdominal muscles in infantile paralysis is rare.

It is a matter of common record that the paralysis at onset is more widespread than at any subsequent time. On this account, coupled with deficient observation, it is possible that

[1] From the Neurological Dispensary, Johns Hopkins Hospital.

many cases showing participation of the abdominal muscles are overlooked. This seems the more probable when we consider the statement made in many text-books that the trunk muscles, though often paralyzed at first, rarely remain so, together with the fact that, especially in dispensary practice, the patients are not seen for some days or weeks after the onset. In the absence then of adequate data on the occurrence of abdominal involvement in the initial paralysis we may provisionally state that if frequent, the tendency in the vast majority of cases is to rapid recovery. The rarity of a residual paralysis in this region may be judged from a consideration of some of the monographs on infantile paralysis.

Seeligmüller (1) in his series of 75 did not have a case of either back or abdominal paralysis.

Sinkler (2) gives in his 340 cases 22 as having involvement of "trunk," none of these, I am informed, included abdominal muscles, but showed impairment only of the muscles of the back, which in every instance was temporary.

Starr (3) in his text-book makes only passing reference to the subject, but gives 4 in his series of 117 cases which showed involvement of "trunk." He believes probably one-third of the trunk cases have paralysis of the abdominal muscles. He has seen 6 such cases, but I am not informed whether they were temporary or permanent. I have been able to collect the following cases which resemble more or less the one I report.

Duchenne fils (4) in his series of 75 cases mentions one a boy of 10 months, who 6 months previously had a generalized paralysis. The movements returned to the upper extremities, but the legs showed a flaccid paralysis in most of the muscles. On the right the muscles of the abdomen and trunk were for the most part atrophied, giving a dorsal scoliosis with convexity to the right. The abdominal walls on the affected side were thin and gave no response to electrical stimulation. When the infant cried there was contraction only on the left, while on the right the abdomen bulged out in a hernia-like prominence. The child was lost sight of and the final outcome was not learned. Duchenne states, "this observation of atrophic paralysis of the muscles of the trunk and abdomen is the only one I know of, and I have not been able to find a similar one in the literature on the subject."

Birdsall (5) has reported one case in which the paralysis was also unilateral.

Bernheim (6) and Leiner (7) have each described one. The former, one of the oblique muscles; the latter, a case very similar to mine, except that the patient was 12 years old.

In the large number of cases of poliomyelitis seen at the Johns Hopkins Hospital Dispensary, the following is the only one, so far as I am aware, showing a residual paralysis of the abdominal muscles. The case lends an additional interest in that it presented the crossed type of paralysis of the extremities, which appears only in a very small proportion of the cases.

Isaac H., Russian Jew; aet. 21 months; admitted to the Johns Hopkins Hospital Dispensary, August 6 (No. C62260). The family history is negative. The child was said to be healthy until present attack. From appearances he must have been surrounded by squalid conditions. Two weeks previous to admission or about July 23, he had an attack of fever lasting three days. During this time the child slept almost constantly. Afterwards it was noticed that the infant could not sit or stand, and that the right leg and left arm were paralyzed. By August 9 the movements had returned to both affected members, the hand to a less degree. By August 20 the child could walk, using both arm and leg fairly well. No note was made on the condition of the abdomen prior to September 15 when the paralysis was first noticed. There must have been asymmetry from the first, which became more apparent as the muscles grew more flaccid and atrophic. November 15. *Present Condition.*—The child is fat and healthy looking, complexion ruddy. Weight 27.5 pounds, height 31 inches, head, occipto-frontal circumference, 19.7 inches (Holt gives 26.5, 32.5, and 18.9 as corresponding figures for a boy of 2). The head is somewhat square-shaped, with undue prominence of frontal and parietal eminences, while the vertex tends to flatness. Chest is well-formed, symmetrical, and expands equally both in quiet and deep breathing. The diaphragm and intercostals are normal. There is no rickety rosary; no bracelets. There are no visible signs of paralysis in arms or legs except the left arm seems slightly smaller and measures 0.5 cm. less than the right, at its middle point. The forearms measure alike. The biceps tendon, radial biceps, ulnar and triceps reflexes are present, equal and normal. The thighs and legs measure alike. The knee-jerks are lively and equal. Tendo Achillis reflex normal. Scratching the soles of his feet produces flexion of all the toes and of the foot, alike on both sides. It is difficult to test accurately the relative muscular strength, but all movements in arms and legs seem symmetrically strong. The infant walks well, but favors the right leg somewhat, rotating it outward so as to step off the inner side of foot.

Abdomen.—The relative hepatic dulness begins 3 cm. above the costal margin and extends 8 cm. downward in nipple line. In mid-line by dipping, it extends 8 cm. below the ensiform bone. Spleen is palpable. Palpation and percussion are otherwise negative. In dorsal decubitus the abdomen is full, but is more prominent on the right. Abdominal skin reflex is present on left, absent on right. Umbilicus is in mid-line. As the child cries and raises the intra-abdominal pressure, the right side balloons out very markedly. On palpation the muscles may be felt to contract under the hand on the left, but not on the right side. In the erect position the conditions are intensified. The left abdomen is now seen to be quite prominent. (Patient's sister states that the belly was quite large before the illness, and we may infer a mild previous rickets.) The asymmetry is much more evident in this position, and as the child cries, the right side balloons out to the full limit of the flaccid and paralyzed wall. Fig. 1 shows this condition in profile. Fig. 2, as well as Fig. 1, shows the greatest protrusion is in the lower right quadrant. Fig. 3 is a profile of the left side, the child is sobbing, which amounts

for the indistinct outlines and gives this side a somewhat fuller appearance than in reality. The paralyzed muscles are the obliqui; the transversalis, and half of the rectus abdominis on the right. An attempt was made to test their electrical reactions, but as the child unfailingly cried every time the current was applied, nothing definite could be learned. As the pictures show, there is no lordosis. There is, however, a slight scoliosis in the lumbar region, with convexity to right.

In conclusion I wish to thank Dr. Thomas, from whose clinic I report this case, and Dr. Cushing for kind help.

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THE DIAGNOSTIC VALUE OF TUBERCULIN IN ORTHOPEDIC SURGERY.

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AND

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For the permanent cure of tuberculosis in any form we must be able to recognize the presence of the disorder while it is still in its incipiency. Any means, therefore, which will afford us an opportunity to attain this end at an earlier date than ordinarily we are accustomed to do, will go far toward lessening the death rate, will increase the percentage of cures, and cannot fail to be of the greatest benefit to mankind.

Koch (1) in 1890 described such an agent in tuberculin, and in his earliest articles (2) definitely stated its diagnostic, as well as its therapeutic value. But amid the enthusiasm of the medical profession over a discovery that produced a specific for the cure of tuberculosis, practically no attention was at first paid to its diagnostic properties. Year by year, however, despite the disfavor into which it speedily fell as a therapeutic agent, tuberculin has gradually pushed its way to the front again until today it is recognized as one of the most important diagnostic agents in beginning tuberculosis.

We do not propose in this paper to speak of its therapeutic or diagnostic properties in incipient pulmonary or glandular tuberculosis. Much has been written on these subjects by Fränkel (3), Mettetal (4), Casselbery (5), Finkler (6), Guttman, Otis (7), Trudeau (8), Tinker (34), and others. Here we would rather refer to the results in a series of cases which, we believe, go to show the great value of tuberculin as a means of diagnosis in bone and joint tuberculosis. Take, for instance, disease of the spine. In very early cases how many of us are there who have not too often been in doubt whether we were dealing with tuberculosis, rachitis, rheumatism, syphilis, spondylitis deformans, or simply a sprain? Is it not sometimes difficult in diseases of the hip to differentiate tuberculosis from synovitis, gonorrhœal arthritis, rheumatism, osteo-myelitis, coxa vara, or fracture? The same may be said in disease of the knee, ankle and other joints. We are fully convinced that in the proper use of tuberculin we have a perfectly harmless method, simple in its application, which will almost invariably tell us of the presence of tuberculosis long before it can be detected by any other means; nay more, tuberculin not only shows us that we are dealing with a tuberculous process, but also in the great majority of instances points out the location of the focus.

The tuberculin used by us came from the laboratory of Dr. Trudeau, to whom we are greatly indebted for a liberal supply. His manner of preparing it is very similar to that of Koch. The tuberculin is a 50 per cent glycerine extract of the bouillon on which the tubercle bacilli have been grown. The flasks are inoculated from a well developed culture of actively growing bacilli, and are then kept in the incubator for six weeks at a temperature of about 37° C. For the tuberculin the contents of flasks are chosen which show a well developed growth over the surface of the bouillon. From one liter of these about 100 cc. of tuberculin are procured. The cultures, being boiled for fifteen minutes, are filtered through paper; the fluid is then put in a porcelain vessel and boiled slowly down to the required concentration (usually 1/10). This concentrated remainder is then passed through a Berkefelder filter. The filter is thoroughly cleansed before and after each filtration and the tuberculin is passed through while it is moist from its last cleansing with sterile water. We have now obtained our stock solution. For injection this solution is mixed with ½ per cent carbolic acid solution, so as to give the strength required and should always be freshly made.

Our mode of applying the test is as follows: The temperature of the patient is taken every two hours for a period of

twenty-four hours preceding the injection. The ordinary hypodermic syringe is used with a needle slightly longer than usual. The needle is inserted deep into the deltoid muscle, thorough aseptic precautions being observed. The puncture wound is covered with sterile gauze, which remains on for twenty-four hours. The temperature is taken every two hours for the ensuing 24-48 hours. If the time selected for the injection be between 8 p. m. and midnight, we can neglect the temperature until 6 a. m. the next morning, as the reaction does not begin until six or eight hours later and the patient need not be disturbed during the night. If possible, the temperature during 24-48 hours preceding the injection should be normal. If, however, pyrexia is present during this period, a positive reaction is still recognizable by means of the local signs. A rise of at least 2° F. is required to constitute a temperature reaction. Using this method, we have injected at their homes forty patients in private practice and from the orthopedic dispensary clinic at the Johns Hopkins Hospital. A synopsis of these cases with their reactions will be found in the following table:

this test has been applied in the wards of the Johns Hopkins Hospital where tuberculin has been employed continuously for the past six years. In looking over the series of cases we find that twenty-five gave a positive and fifteen a negative reaction. Of the twenty-five patients in whose cases a positive reaction was obtained, six were subsequently operated on and all proved to be tuberculous. In eighteen of the remaining nineteen cases the diagnosis has been verified by the subsequent history and response to treatment. The only remaining case, while proven to be tuberculous by its positive reaction to tuberculin, both general and local, is of such recent date that the subsequent history is not as yet available. In regard to the fifteen patients who did not react, one was operated upon and no evidence of tuberculosis was found; of the other fourteen, eight have recovered under non-tuberculous treatment; the remaining six are improved and all suspicion of tuberculosis has been removed. Radiographs were taken of fifteen out of the twenty-five cases which reacted to tuberculin. Of these fifteen cases, six showed a focus, in five the disease was suspected only, and four showed no lesion. The

Case.	Radiograph.	Provisional Diagnosis.	Tuberculin.	Diagnosis.
1. W. F., æt. 1½ years...	No	Potts disease	Mgm. 1.5. Mgm. 3.5, positive	Potts disease
2. W. M., æt. 32 years ...	No	Potts disease	Mgm. 2. Mgm. 4, negative	Syphilitic spine
3. W. M., æt. 16 years ...	No	?	Mgm. 1, negative	Osteo-arthritis spine
4. W. M., æt. 37 years ...	Yes	?	Mgm. 5, negative	Osteo-arthritis spine
5. W. M., æt. 24 years ...	Yes	Potts disease	Mgm. 3. Mgm. 5, negative	Osteo-arthritis spine
6. W. F., æt. 3 years.....	No	Rachitis	Mgm. 2, positive	Potts disease
7. W. M., æt. 8 years	No	Potts disease	Mgm. 2, positive	Potts disease
8. W. M., æt. 25 years ...	No	Scoliosis	Mgm. 2. Mgm. 6, negative	Scoliosis with sciatica
9. W. M., æt. 16 years ...	No	Potts disease	Mgm. 5, negative	Osteo-arthritis spine
10. W. M., æt. 5 years	No	Tuberculosis of hip joint	Mgm. 2, positive	Tuberculosis of hip joint
11. C. M., æt. 10 years....	Yes	Tuberculosis of hip joint	Mgm. 3 and 4, positive	Tuberculosis of hip joint
12. W. F., æt. 12 years....	Yes	Effusion into joint	Mgm. 5, positive	Tuberculosis of hip joint
13. W. M., æt. 3½ years...	Yes	?	Mgm. 3, positive	Tuberculosis of hip joint
14. W. F., æt. 7 years.....	Yes	?	Mgm. 5, positive	Tuberculosis of hip joint
15. W. M., æt. 8 years	Yes	?	Mgm. 5, positive	Tuberculosis of hip joint
16. W. F., æt. 16 years....	Yes	?	Mgm. 3, positive	Tuberculosis of hip joint
17. W. M., æt. 17 years....	No	Effusion into joint	Mgm. 2, negative	Effusion into joint
18. W. F., æt. 29 years....	No	?	Mgm. 2 and 5, negative	Sciatica
19. W. M., æt. 15 years ...	Yes	Fracture	Mgm. 2 and 4, positive	Tuberculosis of hip joint
20. W. M., æt. 12 years ...	Yes	Tuberculosis of hip joint	Mgm. 3 and 5, negative	Coxa-vara
21. W. M., æt. 5 years.....	No	Coxa-vara	Mgm. 1 and 3.5, negative	Coxa-vara
22. W. F., æt. 3 years	No	?	Mgm. 1.5 and 2.5, negative	Synovitis of hip
23. W. F., æt. 32 years....	Yes	Tuberculosis of both hip joints	Mgm. 2, positive	Tuberculosis of both hip joints
24. W. M., æt. 6 years	Yes	.....	Mgm. 3 and 6, negative	Coxa-vara
25. W. F., æt. 43 years....	No	Tuberculosis of hip joint	Mgm. 4, positive	Tuberculosis of hip joint
26. W. M., æt. 12 years ...	No	Tuberculosis of hip joint	Mgm. 3, positive	Tuberculosis of hip joint
27. W. F., æt. 4 years.....	No	Tuberculosis of hip joint	Mgm. 2.5, positive	Tuberculosis of hip joint
28. W. M., æt. 6 years.....	No	Tuberculosis of hip joint	Mgm. .5, positive	Tuberculosis of hip joint
29. W. F., æt. 3 years	Yes	Tuberculosis of hip joint	Mgm. 1, positive	Tuberculosis of hip joint
30. W. F., æt. 30 years....	Yes	Gonorrhœal rheumatism	Mgm. 5, positive	Tuberculosis of hip joint
31. C. M., æt. 9 months...	No	Tuberculosis of knee joint	Mgm. 1.3, positive	Tuberculosis of knee joint
32. W. F., æt. 16 years....	Yes	?	Mgm. 3 and 5, positive	Tuberculosis of knee joint
33. W. M., æt. 10 years ...	Yes	Tuberculosis os knee joint	Mgm. 2.5, positive	Tuberculosis of knee joint
34. W. F., æt. 23 years....	Yes	Chronic synovitis	Mgm. 5, negative	Chronic synovitis
35. W. M., æt. 10 years....	Yes	?	Mgm. 2 and 5, negative	Chronic synovitis
36. W. F., æt. 8 years.....	Yes	Tuberculosis of ankle joint	Mgm. 5, positive	Tuberculosis of ankle joint
37. W. F., æt. 4 years.....	Yes	Tuberculosis of ankle joint	Mgm. 2, positive	Tuberculosis of ankle joint
38. W. F., æt. 20 years....	Yes	Old fracture os calcis	Mgm. 4, negative	Old fracture os calcis
39. W. M., æt 2 years.....	Yes	Green stick fracture tibia	Mgm. 3, positive	Tuberculosis of lower end of tibia
40. W. F., æt. 21 years....	Yes	Tuberculosis of ankle joint	Mgm. 3, positive	Tuberculosis of ankle joint

These cases were selected almost invariably in order to clear up the diagnosis. Our results show the ease with which tuberculin may be used in private practice and the accurate data that can be obtained under such circumstances. We have not included here the large number of cases in which reasons for this will be discussed later. For convenience we have arranged the patients in groups according to the joint involved. A few cases of especial interest will now be discussed briefly.

Case 1. White; female; aged one year and six months;

admitted to the clinic January 10, 1900. Three days before she had a slight fall. Although she had previously walked for four months she now prefers to crawl; cries and is fretful. Examination showed only a slight restriction to hyperextension of the back; no kyphosis. Seat of pain not to be located. Two physicians connected with the dispensary had seen her just before admission, one suggested rachitis of the spine; the other malnutrition. Two injections of tuberculin were given. The first (1.5 mgm.) was followed by a rise of temperature to 100° F. The reaction was considered suggestive. A few days later 3.5 mgm. were given, and seventeen hours later the temperature was 100.5° F., having been normal previous to the injection. The child refused nourishment, and was very restless. On examination of the back, the resistance to hyperextension was greatly increased; the child refused to stand; the knee-jerks were increased. Here then was a positive reaction both general and local. On the following day the temperature had returned to normal and the general and local signs were exactly as those which had been present previous to the injection. The child was treated for Pott's disease with plaster jackets and a Taylor back brace. The note of her condition three years after the beginning of treatment is as follows: "The child is practically well with a slight kyphosis of the third lumbar vertebra, having passed through a typical Pott's disease during this period."

In this case tuberculin gave us a positive diagnosis at a time when the clinical symptoms were not sufficient to warrant a positive decision, and allowed treatment to be instituted in time to prevent deformity.

On the other hand, the negative value of tuberculin is shown in the following case:

CASE 2. White; male; aged thirty-two; admitted to orthopedic clinic October 5, 1901. Referred by the nervous clinic with a diagnosis of tuberculosis of the spine. *Family history*, negative. *Previous history.* Lues ten years ago. *Present illness.* The patient has complained of pains in his back radiating to the abdomen for the past five months. The pains are present during the day and night. The back is becoming stiff in the lumbar region, and the patient finds it difficult to pick up objects from the floor; there has been a continuous loss of strength, and the man is not steady on his feet. Loss of weight, fifteen pounds. *Examination:* The patient walks with an unsteady gait; holds his back perfectly rigid. There is some obliteration of the lumbar curve, but no actual kyphosis. The knee-jerks are markedly increased. Ankle clonus is present. There is considerable pain attending a jarring of the heel. A probable diagnosis of tuberculosis of the spine was made and the patient was treated by means of plaster jackets for the first few weeks. He thought he improved a little; but later the pain grew worse at night. The symptoms of paralysis increased and the man lost weight rapidly. For six weeks we persisted in the treatment, but the patient gradually grew worse. On December 28, 1901, he was given 2 mgm. of tuberculin. There was no general or local reaction. On December 30, 4 mgm. were given. No reaction followed, either general or local. This caused us to change our diagnosis and for the first time to think seriously of his specific history as an etiological factor. Treatment with inunctions and potassium iodide internally was then begun. Within a month the patient gained three pounds in weight; the pain ceased almost entirely; no ankle clonus could be obtained and his knee-jerks became far less lively. He continued to wear a laced plaster jacket as a means of support. On May 1, 1902, four months after the initiation of the syphilitic treatment, he felt perfectly well; no brace was worn; the back was not stiff; all symptoms had disappeared. Fifteen months after beginning the treatment, he reported for observation and seemed well in every respect. Specific treatment, however, was continued. This case shows the value of tuberculin in eliminating tuberculosis and arriving at the true etiological factor.

Again let us contrast two cases of disease of the hip-joint.

CASE 13. Boy; white; age 3½ years; admitted to the orthopedic clinic March 3, 1903, complaining of a slight limp. Mother died of pulmonary tuberculosis. He had had diphtheria two years previously. *Present sickness.* Four months ago he had a slight fall on the floor while playing and since that time has had a limp which has slightly increased. There are no other subjective symptoms. Examination shows a slight limp. There is an apparent shortening of ½ cm. No other deformity. Possible flexion of 85° and possible abduction of 30°. No resistance to hyperextension, adduction, internal or external rotation. No muscle spasm. Very slight atrophy of thigh and calf.

In this case the diagnosis was not plain; it was by no means a well defined case of tuberculosis of the hip. The radiograph showed that the distance from the trochanter to the head of the femur was slightly less than on the other side. There appeared to be a slight roughness of the acetabulum and the trochanter possibly showed a slight rarefaction.

On March 25, 1903, at the patient's home, 3 mgm. of tuberculin were given in the usual manner. Fourteen hours afterward the temperature had reached 101° F. This was accompanied by loss of appetite and extreme restlessness. All the local signs were decidedly increased. Pain in the hip and knee was complained of. There was distinct muscle spasm. The leg assumed a position of slight flexion and there was a decided limitation of all the motions. On the following day the hip had returned to its former condition and the general symptoms had disappeared. Here the reaction was positive. The radiograph, owing to the age of the child, did not show a definite focus, but under the influence of tuberculin the local signs presented such a definite picture that there could be no doubt as to the tuberculous nature of the affection.

The absence of a positive reaction on the other hand in diseases of the hip of a non-tuberculous character is shown in the following case:

CASE 20. White; male; aged 12 years; admitted May, 1902, complaining of a limp. Family and previous history unimportant. *Present illness.* In October, 1901, the boy was hurt on the thigh while playing foot-ball and began to limp. There was no history of night cries and no pain. He

was put up in a plaster spica and given crutches, which he used for six weeks. A tentative diagnosis of hip disease was made by a prominent surgeon. After the cast was removed he continued to limp. He was seen by us in May, 1902, six months after the original injury, and on examination the leg showed marked outward rotation, slight apparent shortening. Flexion was limited to 60°, abduction to 45°, and no rotation was allowed. No pain and no muscle spasm. The diagnosis was made of slight coxa vara, non-tuberculous in origin. Three and five mgm. of tuberculin were given at an interval of two days with absolutely no local or general reaction. The diagnosis of coxa vara was thus confirmed by a negative reaction to tuberculin and a radiograph, which showed no tuberculous focus, but a slight bending of the neck of the bone. The treatment consisted in massage and passive motion, both of which would have been contra-indicated had the joint been tuberculous. It is now eleven months since he was first seen by us. There is very little limp; the possible flexion is now 90°, possible abduction 80°.

The last case that we will discuss in detail seems to be of particular importance:

Case 32. White; female; aged 16 years, admitted, February, 1903, complaining of pain in the left knee on pressure and while standing. *Family history.* Father and mother both dead of pulmonary tuberculosis. Previous history, unimportant. *Present sickness.* Three weeks ago she had a slight fall while skating, to which no attention was paid. Since that time there has been a slight limp. The only symptom she presents is pain on pressure over the patella. There are no other local signs of disease. On February 3, 1903, she was given 3 mgm. of tuberculin. Eighteen hours afterwards the temperature had reached its maximum of 100.8° F., after which it rapidly fell to normal. There were general symptoms of nausea, headache and loss of appetite. Of greater importance we think were the local signs, for, whereas before the injection, there was only pain on pressure over the patella, there was now a slight rise of local temperature; the motions were restricted in extreme flexion and extension, and even the slightest pressure over the patella would cause exquisite pain. On the following day the temperature was normal; the general symptoms had disappeared and there remained locally only the slight pain as before the injection. The radiograph showed a slight focus of disease, about 1½ cm. in length and ½ cm. broad, situated in the internal condyle, running toward the joint. The tuberculous nature of the disease having been so early established by means of the tuberculin, and its extent ascertained by radiography, it was decided to see if the focus could not be removed and a stiff joint avoided. Accordingly the joint was opened for inspection, and on the top of the internal condyle directly under the patella a circumscribed raised area of inflammation was found about 1½ cm. in diameter, which showed, however, no tubercles to the naked eye. This area was left alone, the joint was closed, the area of diseased bone immediately beneath this seat of inflammation was removed (extra-articular) and proved to be tuberculous. The leg was then put in plaster. Three weeks later pressure over the patella elicited no pain and the motions of the joint were found to be good.

This case illustrated exceptionally well the diagnostic value of tuberculin in the earlier cases of tuberculosis. In this instance its employment rendered operative interference possible without the destruction of the function of the joint.

As a rule, when tuberculin is given, definite general and local signs are present whenever tuberculosis is present, and are generally absent whenever tuberculosis is absent. The smallest focus of tuberculosis will give a positive reaction and hence the diagnosis can be cleared up at a much earlier date than is possible by any other means.

During the twelve years that tuberculin has been used the dosage has varied with different observers. This in itself accounts for the failure in numerous instances to detect the presence of tuberculosis. Such small doses were used that small foci did not give either a general or a local reaction. Grasset (9) says that 2/10 to 5/10 mgm. are useful, while White (10) reports that doses of 1 to 2 mgm. are not to be relied upon. Mettetal (4), using tuberculin in children, gives doses from .1 to .5 mgm. On the other hand, if the dose be too large, even normal individuals may react. According to Koch (11), 10 mgm. may cause such a reaction. He obtained a reaction in himself with a dose of 25 mgm. Often such small doses were given in quick succession that a tolerance was created against a larger dose. So varied are the factors on which the dosage depends, the age of the patient, the seat of the focus, the strength of the tuberculin, its deterioration, and individual susceptibility, that no hard and fast rule can be given. Madison (12) in his recent article follows an initial dose of 4 mgm. by 7 to 10 mgm. when necessary. It has been our custom in small children to give an initial dose of ½ mgm. followed at intervals of two days with 2 two 4 mgm., when necessary. In older patients, 2 mgm. are used as an initial dose, followed in two days by 4 and 6 mgm. as a maximum. In no case have we found it necessary to give more than 6 mgm.

As to what constitutes a positive temperature reaction, all observers are agreed in requiring a rise of at least 2° F. The initial rise generally takes place within the first twenty-four hours and the pyrexia usually lasts over a period of twenty-four hours. The temperature may rise even as high as 104° or 105° F.; the curve of the pyrexia may be atypical, being prolonged over the usual twenty-four for forty-eight hours or more; or the time of the onset may be delayed, but this is due no doubt in most part, as claimed by Madison (12), to deterioration of the tuberculin.

A definite group of symptoms always accompanies the fever; headache, anorexia, nausea, occasional vomiting, restlessness, etc. To those, however, who are dealing with joint and bone tuberculosis the local symptoms are of far more interest and of equal importance. Previous to the injection of tuberculin the local seat of disease should be examined carefully for all signs of the suspected trouble, such as the permanent deformity, possible motions, muscle spasm, local temperature and pain. In response to the tuberculin, if

tuberculosis be present, we shall have all the local signs of the disease distinctly increased, that is, motion will be more limited, deformity will be greater, tenderness and muscle spasm will be exaggerated, and local temperature may be increased; in fact, the local signs alone are so characteristic of a tuberculous process that the diagnosis can be made from these alone.

Koch (1) and Maragliano (13) have laid stress upon the local signs observed in lupus, glandular and pulmonary tuberculosis. Klebs (14) mentions local reactions occurring in disease of the bones and joints, while Fränkel (3) says, "Tuberculin is especially useful in surgical tuberculosis, particularly as in such cases the local phenomena can be so easily observed." On the other hand, Frazier and Biggs (15) are the only observers, so far as we know, who claim an entire absence of a local reaction in tuberculosis of bones and joints. We can only say that in every case that has come under our personal observation the local phenomena, where a positive reaction was obtained, have been of a most pronounced character. The selective action of tuberculin for tuberculous tissue is well recognized, and whenever a patient with a suspected joint gives a general but no local reaction, the probabilities are strongly in favor of the view that the joint in question is non-tuberculous. It is evident, therefore, that the local signs should be carefully observed, as well as those of a general character, in order to definitely locate the tuberculous process.

Is the use of tuberculin harmful in its effects?

A few months after its discovery, Virchow stated that in his opinion it might be harmful. This view has been concurred in by Ambler (16) and Knopf (17) on theoretical grounds. Head (18), Heron (19) and Ewald (20) have reported cases in which very weak patients have died after a large injection; but the evidence is by no means conclusive that death was due to the injection.

On the other hand, the great majority of observers, among whom may be mentioned Fränkel, Mettetal, White, Lathan (21), Neff (22), Osler (23), Klebs, Martin and Robbins (24), France (25), Elder (26), and Voges (27), are unanimous in their statements that, when tuberculin is used in proper doses, there are no harmful effects. We have never seen a case in which any permanent damage had been done by tuberculin. A dose which is too large may produce a fever that is prolonged beyond the usual twenty-four hours, and the local signs may persist for a longer time than usual, but within a day or two the temperature always returns to normal and the local signs become similar to those before the injection.

Is tuberculin a specific in the diagnosis of tuberculosis?

According to some observers, syphilis, leprosy and actinomycosis react to tuberculin. Mettetal (4), Billroth (28) and von Eiselsberg (29) have seen cases of actinomycosis which gave a positive reaction, but Mettetal definitely states that the reaction was atypical. Goldschmidt (30), Babes and Kalendro (31) state that leprosy may react. In 109 autopsies of lepra reported by Abraham (32), 30 per cent showed tuberculous lesions. It seems highly probable, therefore, that most of the reactions reported in leprosy were due to an accompanying tuberculosis. In regard to syphilis, Beck (33) states that one-half of such cases react to tuberculin, while Otis (7) in 35 cases in which the diagnosis was carefully made, found only 12 per cent that reacted, and he considers Beck's figures as entirely too high. Fränkel, on the other hand, thinks the probability of obtaining reactions in uncomplicated cases of syphilis very doubtful. He reports a case of a syphilitic ulcer, which gave a general, but no local reaction to tuberculin. The ulcer was cured by inunctions and potassium iodide internally, and again the patient gave a general reaction to a dose of 1 mgm. He considers that there he was dealing with syphilis complicated by hidden tuberculosis.

The only patient in our series (Case 2) in whose case a reaction to syphilis was in question, failed to react to 2 and 4 mgm. of tuberculin and entirely recovered under specific treatment. From the cases of syphilis reported by various observers, it seems probable that a small number give a reaction, but further evidence is necessary to show that there is no associated tuberculosis in these cases. Broadly speaking then, it may be said that tuberculin is practically a specific in the diagnosis of tuberculosis.

The radiograph has come to play an enormous role in the work of the orthopedic surgeon. It is one of the most helpful means which we possess to point out lesions of bones and joints and yet it has its limitations, especially in the very cases in which we most need its help. By far the greater number of our patients with bone and joint tuberculosis are children. The structure of their bones and the relatively large proportion of cartilage in such cases does not lend itself to differentiation by means of radiography as readily as in adults. It has often been our experience in children that even when the clinical signs were sufficient to make a diagnosis of tuberculosis, the best radiograph would fail to present a picture of the disease process or would do it so imperfectly as to make us doubt its existence. In such cases tuberculin would invariably give us both a general and local reaction. On the other hand, tuberculin plus the radiograph forms a combination of diagnostic agents which are of the greatest value, especially in adults, as is shown in case 32, where tuberculin gave the diagnosis and the radiograph showed the location and extent of the focus.

The application of the tuberculin test is in reality a most simple procedure, much easier than a blood count and no more difficult than an ordinary injection. Naturally, the method is more accurate in hospital work, owing to the aid of a competent nursing staff, and yet with a little more exertion on the part of the physician, private and dispensary cases need but little more care. The same physician who examines the case before injection should also do so at the height of the reaction. The temperature must be carefully taken and recorded every two hours. It is generally agreed that tuberculin is an unstable product and deteriorates if not kept in a cool, dark place. The ½ per cent carbolic acid solution should be made up freshly before each injection. The majority of observers

agree that it may be used if not kept more than three days. Madison (12) believes that a fresh bottle of the stock solution should be opened once a month. In his opinion the failure of tuberculous cases to react and delayed reactions are primarily due to deterioration of the tuberculin.

The reliability and harmlessness of the test may be judged by the immense number of observations upon cattle where autopsies can be obtained and results verified. Voges (27) in eight thousand autopsies upon cattle has shown that the test is accurate in 90 per cent. Koch, with an experience of three thousand cases, considers the test almost absolute.

Our own work, when taken in conjnction with that of others, would appear to justify the following conclusions:

1. Tuberculin is the best and most reliable diagnostic agent for incipient tuberculosis of bones and joints.

2. Its proper administration is attended by no permanent harmful effects.

3. The dosage is variable and it is rarely necessary to exceed a dose of 6 milligrams.

4. The local signs are of equal, if not greater, importance than the general reaction, in bone and joint tuberculosis.

5. Tuberculosis practically always reacts to tuberculin.

6. Diseases other than tuberculosis may possibly react to tuberculin, but the evidence on this point is not conclusive.

7. The diagnosis of tuberculosis can be made earlier and with more certainty by means of tuberculin than by radiography.

8. The tuberculin test is applicable to private and dispensary as well as to hospital practice.

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28. Billroth: Quoted by Knopf: Jour. Am. Assn., Dec. 9, 1899, Vol. XXXIII, No. 24, p. 1446.

29. von Eiselberg: Quoted by Knopf: Jour. Am. Assn., Dec. 9, 1899, Vol. XXXIII, No. 24, p. 1446.

30. Goldschmidt: Berl. Klin. Wchnschr., 1891, p. 28.

31. Babes and Kalendro: Deutsche Med. Wchnschr., 1891, p. 115.

32. Abraham: Allbutt's System of Medicine, Vol. III, p. 59.

33. Beck: Deutsche Med. Wchnschr., Nr. 9, 1899, p. 137.

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INSTRUCTION IN PSYCHIATRY AND NEUROPATHOLOGY.

A limited number of graduates in medicine can have an opportunity for work in the laboratory of the Sheppard and Enoch Pratt Hospital.

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For particulars, apply by letter to Dr. E. N. Brush, Physician-in-Chief and Superintendent, Sheppard and Enoch Pratt Hospital, Station "A," Baltimore.

A PRACTICAL METHOD OF DEMONSTRATION.

BY W. J. CALVERT, M. D.,
University of Missouri, Columbia, Mo.

The problem of demonstrating either the normal or abnormal relations of organs has presented many difficulties which have given rise to many devices such as serial sections of the body, schematic drawings, models, etc., all of which are of value, but none of which are entirely satisfactory. In addition many of the larger drawings and most models are expensive and are beyond the reach of many institutions and most individual students.

Wherever practicable full sized drawings are the most desirable as the student then is not required to mentally convert a one-fourth, one-half or a three-fourth sized drawings to one of full size.

For full sized outline drawings the tracing-cloth and blue-print method extensively used by architects and engineers is very satisfactory. Four original drawings are necessary. One each of the anterior, posterior, right and left sides with the outlines of the several organs projected on each are sufficient. From these, full sized outlines of the thorax, abdomen or pelvis, as desired, may be traced in a relatively short time and the outlines of an abnormally placed organ, etc., may be traced in at will. When the tracing-cloth outline is completed, blue-prints can be made for a few cents each by men who are to be found in every city. In this way as many drawings as is desired may be made and each member of a class can afford to buy a blue-print, which, when worn out, may be replaced at little expense.

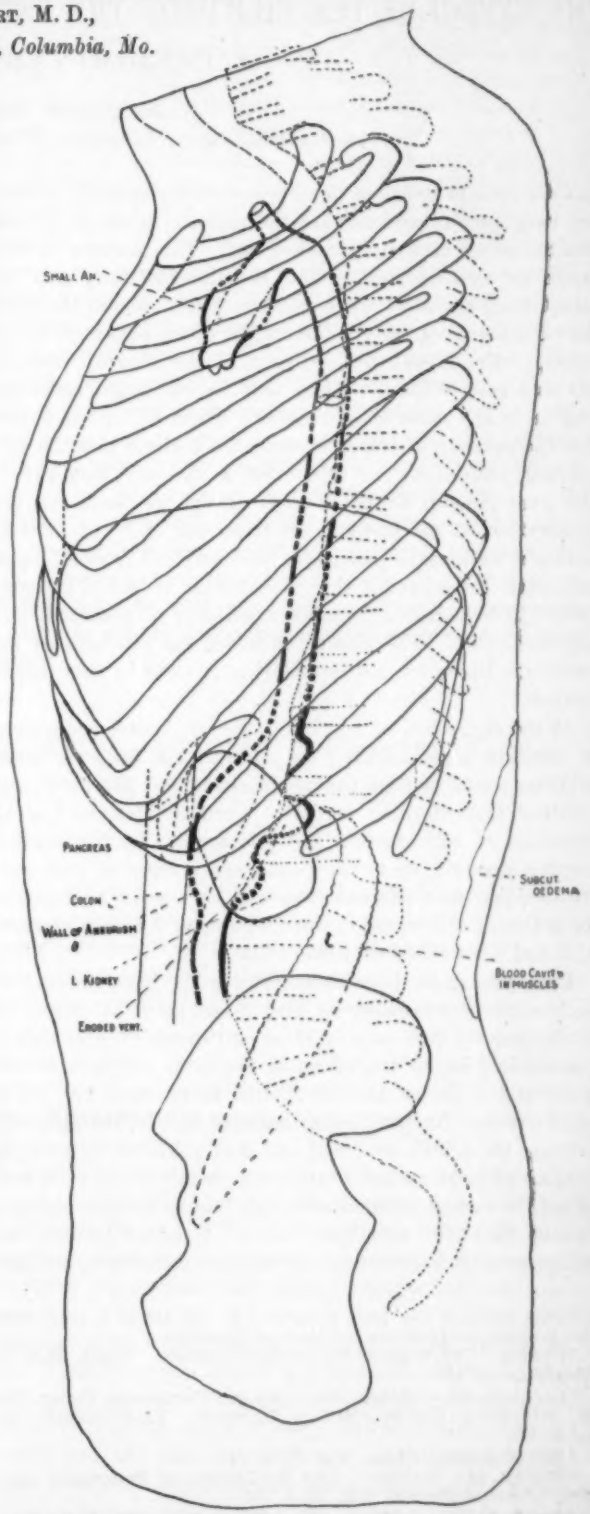
One distinct advantage is that the drawings are not schematic, but are as accurate as measurements allow. In addition they are easily made, cheap, and can be replaced when worn out. They are of especial advantage for demonstrating individual cases to societies, classes and for lecture work.

The materials required are a drawing board, a few thumb tacks, tracing-cloth, india ink and ordinary pens.

If desired, the outlines of the bones or organs may be colored or may be blackened with a soft lead pencil which can be erased later.

From the drawings on cloth, photographs may be easily made for reproduction.

For the original outline drawing of the print which illustrates this article I am indebted to Dr. Potter of the St. Louis University.



Reduced from life size. Heavy black interrupted line indicates lesions.

THE JOHNS HOPKINS HOSPITAL BULLETIN.

The Hospital Bulletin contains details of hospital and dispensary practice, abstracts of papers read, and other proceedings of the Medical Society of the Hospital, reports of lectures, and other matters of general interest in connection with the work of the Hospital. It is issued monthly.

Volume XV is now completed. The subscription price is $2.00 per year. The set of fifteen volumes will be sold for $75.00.

THE EFFECT OF THE BILE UPON THE ESTER-SPLITTING ACTION OF PANCREATIC JUICE.[1]

A PRELIMINARY COMMUNICATION.

By Albion Walter Hewlett, M. D.

(From the Physiological Laboratory of the Cooper Medical College, San Francisco, Cal.)

That the bile assists in the digestion and absorption of fats has long been recognized, but the manner in which it does this has never been fully worked out. The presence of bile favors the emulsification of fats and this indirectly aids the fat-splitting action of the pancreatic juice, because the finer the emulsion the greater is the surface of fat presented to the action of the fat-splitting enzyme. Little regard, however, has been paid to the possibility that the bile might assist the enzyme in any more direct manner. Pawlow[2] indeed asserts that the presence of bile so changes the medium that the fat-splitting proceeds two or three times as fast as it would if no bile were present. Bruno, a pupil of Pawlow, estimates the acceleration of the lipolytic action as ten to twenty times.[3] Glassner, working on pancreatic juice obtained from a human pancreatic fistula, found that the addition of bile to the pancreatic juice increased its action upon olive oil about one and one-half times.[4] The manner in which the bile assisted the pancreatic lipase was not especially considered by these investigators.

At the suggestion of Dr. W. E. Garrey, I have endeavored to ascertain more definitely the effect of bile upon the ester-splitting action of pure pancreatic secretion. The latter was obtained from dogs by means of secretin injections[5] or by injections of both secretin and pilocarpin. In this manner varying amounts, up to forty cubic centimeters, of pure pancreatic juice were obtained from each of a series of dogs and the action of this secretion was tested upon a variety of esters with and without the addition of bile.

Pure pancreatic juice decomposes ethyl butyrate surprisingly slowly if we compare it with the action of pancreatic extracts upon the same ester.[6] If one or two cubic centimeters of pancreatic juice be diluted up to five cubic centimeters with water and if the mixture be allowed to act upon 0.25 cc. of ethyl butyrate for twenty-four hours at 37° C., the difference between the acidity produced and that produced by an equal amount of boiled pancreatic juice is usually about 0.1-0.2 cc. of a 1/20 normal solution, although it may in some instances run up to 1.0 cc. or slightly more. Unboiled bile also decomposes ethyl butyrate very slowly, not differing appreciably in this respect from boiled bile. If the bile be added to the pancreatic juice, however, the cleavage of the ethyl butyrate proceeds with great rapidity and in twenty-four hours an acidity amounting to 7.0-16.0 cc. 1/20 normal solution will be produced. Similar results were obtained when ethyl acetate or amyl acetate were used in place of ethyl butyrate.

The ordinary fats are insoluble in water and they are therefore but little suited to a solution of the problem at hand, because any acceleration in the action of the pancreatic juice, which the bile would cause, might be attributed to its emulsifying action. This difficulty however, is, overcome if a soluble triglyceride, such as triacetin, be used, for then the question as to the degree of emulsification is eliminated. It was found that pure pancreatic juice, even without the addition of bile, splits triacetin much more readily than it does ethyl butyrate, ethyl acetate, or amyl acetate. In one experiment the acidity produced in an hour at 20° C. amounted to 0.5 cc. 1/20 normal solution and in twenty-four hours to 12.6 cc. On the addition of bile, the decomposition proceeded at a much more rapid rate just as it did in the case of ethyl butyrate. The acidity produced in one hour amounted to 13.0 cc. and in twenty-four hours to 18.6 cc. The acceleration, therefore, was very marked during the first hour, and the reason that this acceleration was less apparent after twenty-four hours probably lay in the fact that the reaction was approaching an equilibrium.

The addition of bile also accelerates the action of the pancreatic secretion upon emulsions of olive oil, but to what extent and why this occurs has not as yet been fully determined.

Which constituent of the bile possesses this peculiar property of accelerating the ester-splitting action of the pancreatic juice? Boiling the bile does not destroy this property and we may therefore infer that it is not due to an enzyme. Furthermore, this property resides neither in the cholesterin, in the pigments, in variations in reaction, nor in variations in the amount of calcium salts present. Precisely the same accelerating effects may, however, be produced by the addition of lecithin to the pancreatic juice. Thus in one experiment the pure pancreatic juice by its action on triacetin for twenty-four hours produced an acidity amounting to 4.3 cc. 1/20 normal solution; the same plus two cubic centimeters of bile produced 19.5 cc. acidity, and plus two drops of a strong alcoholic solution of Merck's lecithin produced an acidity of 19.9 cc. A commercial preparation of the bile salts will also accelerate the ester-splitting of the pancreatic juice, but it seems that the more the bile salts are purified, the less effect they have in this respect, so that it is possible

[1] This research has been completed by the aid of a grant from the Rockefeller Institute for Medical Research.

[2] Pawlow: The Work of the Digestive Glands. Trans. by W. H. Thompson, p. 159.

[3] Die Galle als wichtiges Agent bei der Verdauung. Inaug. Diss. St. Petersburg, 1897-98. Ref. in Jahresber. Thier-Chemie. Vol. 27, p. 441.

[4] Ztft. f. physiol. Chem., Vol. 40, p. 465.

[5] Bayliss and Starling: The Mechanism of Pancreatic Secretion. Jour. of Physiol., Vol. 28, p. 325.

[6] Kastle and Loevenhart: Am. Chem. Jour., Vol. 24, p. 491.

that the action of the crude preparation is due to a contamination with lecithin.

Various possibilities present themselves as to the manner in which the bile assists the ester-splitting action of the pancreatic juice. In the first place, does it act by converting a proferment into a ferment, an action analogous to that exerted by the enterokinase of the intestines upon the proferment of trypsin?[1] In considering this question, we must remember that if this hypothesis were true, a small amount of bile would be just as effective as a larger amount provided it acted for a sufficiently long time, and that if sufficiently small amounts of bile were added to the pancreatic juice the rate of cleavage could be shown to progressively increase as more and more of the proferment were converted into the ferment. It was found as a matter of fact that these conditions are not realized in the case of the action of bile upon the pancreatic juice. Another possibility is that there is a substance in the pancreatic juice, itself inert, which combines with some constituent in the bile to form the fat-splitting enzyme. Such a condition would be analogous to that described by Kyes[2] for cobra venom. Alone this exerts little if any hæmolytic action; plus lecithin the action is most marked. The pancreatic juice, however, is moderately active even without the addition of bile. It would, therefore, be necessary to suppose that the pancreatic juice normally contains a trace of lecithin, but that this is not sufficient to render active any considerable portion of the fat-splitting ferment. It seems to us more probable that the bile acts as an accelerator upon the fat-splitting ferment, and indeed this is more in accord with the modern view that all ferment action is in the nature of an acceleration. We know that the esters will slowly decompose in pure water and the same is true if they are placed in boiled pancreatic juice. If the secretion be unboiled, *i. e.*, if the ferment be present, the decomposition proceeds at a somewhat more rapid rate, and if to this, bile be added, the decomposition is still further accelerated. We do not know exactly how this last acceleration is brought about, although other similar accelerations are known to us. Thus hydrogen dioxide will slowly decompose in water. If ferric sulphate be added to the water, the decomposition is moderately accelerated. Cupric sulphate alone produces little if any effect upon the hydrogen dioxide, but if it be added to the mixture of hydrogen dioxide and ferric sulphate the reaction is enormously accelerated. Substances which act in this manner have been termed zymoexcitors. It seems probable, therefore, that the bile increases the ester-splitting action of the pancreatic juice by virtue of a zymoexcitor which it contains and that this zymoexcitor is in part, at least, lecithin.

[1] Bayliss and Starling: The Proteolytic Activities of the Pancreatic Juice. Jour. of Physiol., Vol. 30, p. 61.

[2] Berl. klin. Wochenschr., 1902, Nos. 38-39.

A CASE OF RIGHT-SIDED INFANTILE HEMIPLEGIA, WITH A DESCRIPTION OF THE PATHOLOGICAL CHANGES FOUND IN THE BRAIN AND THE SPINAL CORD.

BY ROBERT REULING, M. D., *Baltimore, Md.*

Although the literature of infantile cerebral palsies and their complicated symptomatology is voluminous and their pathology at the present time has at least a good working foundation, it is nevertheless surprising how comparatively few instances have been reported where the brain and spinal cord in these affections have been studied in their entirety. For this reason the following case is presented, inasmuch as a complete examination of the central nervous tissues was possible; unfortunately a clinical history and a satisfactory neurological examination were not obtained.

Sensory changes, however, are of rare occurrence in similar cases; in the present with exception of certain occasional pains in the affected members, a typical example of right-sided hemiplegia with marked contractures and osseous changes, due to its long standing (25 years), little was ascertained.

E. M., single, aged 28, white, born in the United States, was admitted to the lying-in wards of the University of Maryland Hospital in 1900. She complained of severe abdominal pains, and weakness. An abortion had been accomplished a few days preceding her entrance to the hospital. For the following notes and the use of the material I am much indebted to Drs. Alan and Stokes.

It was evident at the first examination that the patient had a septic infection. All means to combat this were tried; including irrigations. Her temperature ranged high and fairly regular for three days after admission, then became irregular, rigors were noted, and delirium of an unusually violent type soon set in. On the fifth day a soft systolic murmur appeared, best heard at the apex; the following day a very faint friction over the body of the heart suggested pericarditis. Soon a præsystolic murmur appeared, all pointing to a septic endocarditis. The abdomen was fairly distended and very sensitive in its lower quadrants.

The uterine discharge was ill-smelling and characteristic of septic endometritis. A few minute petechial hemorrhages appeared in the skin on the chest and abdomen. The urine throughout contained a considerable amount of albumen and casts; later some blood. She died on the tenth day after admission.

Clinical Notes.—Nothing is known as to her family history. When two years old she was taken ill with convulsions and on their cessation the right-sided paralysis was evident. Syphilis in later life could be fairly well excluded. She was immoral and had been the mother of one illegitimate child born at term. Epileptic convulsions have occurred from childhood, at what age they began is not known; they have been

less frequent since maturity. While in the hospital she had two severe general convulsions of an epileptic type, with frothy sputum. Her speech was considerably affected, a distinct hesitancy and lack of motor innervation was noted by all that saw her during the first days of her illness; as to the existence of a sensory aphasia this remains unproven. No athetoid movements were noted.

Autopsy.—Thirty hours after death. Body of moderately well nourished woman; little subcutaneous fat. Rigor mortis present. Abdomen moderately distended and highly tympanitic; skin over the same has a glossy appearance and the umbilicus is flattened. Faint bluish discoloration in left iliac region. Cornea in good condition and pupils regular. No external signs of advanced decomposition. A few petechial hemorrhages in lower chest and on the abdomen. A small linear scar over the right eye. Tongue very dry and covered with sordes, no scars found. No apparent facial asymmetry.

There is a striking difference in the appearance of the right and left upper extremities. The muscles of the right shoulder girdle and the entire right upper extremity are markedly wasted, the biceps muscle is practically a thick fibrous band, the humerus is also atrophied. The right arm is strongly flexed against the side of the chest and the forearm across it, the hand with its fingers flexed into the palm and indenting its surface, lies near the mid-body line over the xyphoid cartilage. Unless adhesions and muscle were torn the extremity could not be placed to the side of the body. An average difference of from 2.5 to 3 cm. is found in measuring the circumference of the right arm and forearm in comparison with the left extremity. The right lower extremity is also atrophied throughout, less markedly so than its upper fellow, it lies in a natural position, excepting that the foot is in a rigid, talipes equinus position, points sharply downwards while the toes are drawn dorsalwards. The tendo achillis was tense. The atrophy of muscle tissue seems most pronounced in the tibialis anticus and gastrocnemius. A shortening of 6 cm. in the right lower extremity was found and in approximating the two feet the toes of the right foot touched only the left internal malleolus.

On making the usual longitudinal incision a considerable amount of ill-smelling gas escapes from the abdominal cavity and on moving the intestines gurgling sounds are heard. An intense acute fibrino-purulent peritonitis, with occasional hemorrhages exists; the adhesions can be broken readily however. Pus is present between a few coils of the smaller intestines more being found in the pelvis where also a mucopurulent, slightly blood-stained fluid exists. The uterus is about the size of the organ at the 4th month of gestation, its muscle tissue is soft and friable. The perimetrium is injected. Uterine vessels are thrombosed, especially the veins. The pelvic glands are much enlarged. On section a raised patch about the size of a dollar is seen in the left half of the uterine endometrium, presenting a beautiful example of so-called diphtheritic inflammation. The kidneys show an acute parenchymatous nephritis and thrombi in many vessels. The liver is about normal size, and shows an acute parenchymatous necrosis. The spleen is moderately enlarged, very soft and vascular, the splenic corpuscles being obliterated. The heart shows its pericardial surface covered with a thin fibrino-purulent exudate the surface being still moist; the pericardial vessels are injected. A mass of thin, fresh adhesions attach almost the entire pericardial surface to the chest and lung pleura. A small amount of cloudy yellow fluid is in the sac. The heart is slightly dilated, its muscle is soft. On section a striking example of acute fibrinous exudate covers the leaflets of the mitral valve; this is raised above their surface and small fragments break off on handling the organ; the exudate extends slightly over to the endocardium and to the attachment of the papillary muscles. About the aortic valve a similar acute inflammation is in its incipiency. The lungs have a moderate pleuritic inflammation on the left side, especially near the cardiac region, also several patches of broncho-pneumonia and numerous emboli in the pulmonary vessels.

Description of the Brain.—On removing the calvarium, one is struck by the marked asymmetry of the two hemispheres; the left being much smaller than the right, this being true of the left hemisphere throughout. On removing the brain it is evident that the entire skull cavity is smaller than normal and the brain below the average size. The left anterior and middle fossæ of the skull are considerably smaller and more shallow than those on the right side, this being especially true of the middle fossæ. The posterior fossæ show, however, a reverse condition in that the right is smaller and more shallow than the left; the reason for this will be clear after considering the further examination of the brain. The dura mater over almost the entire left motor region was of a milky color, extending over the lower posterior central gyri and anterior occipital lobe. This membrane is much thickened and shows evidence of an increase in its vascular supply; in the region of the first, second, and third frontal convolutions a considerable retraction of the dura occurred amounting to the formation of a distinct cavity over the third frontal gyrus. When the removal of this cloudy portion of the dura was attempted, it proved intimately adherent to the brain surface.

Three small cysts were opened from which clear straw-colored fluid escaped, two in the first frontal and one in the second frontal regions. The dura and other membranes appeared about normal over the remaining brain surface. At the longitudinal fissure the left hemisphere showed a retraction of about 1.5 cm. below the level of the upper border of the right hemisphere while its anterior frontal tip was retracted an equal distance from its fellow on the right hemisphere. The only apparent change in the gross specimen of the cerebellum was an atrophy of the right hemisphere in its entirety, the left being normal. This atrophy was evident at first glance. There were no localized retractions of cerebellar tissue. The entire left half of the medulla showed a well-marked atrophy. The right half of the spinal cord below the decussation of the pyramidal tract was also atrophied. In the cord this atrophy was visible in the gross

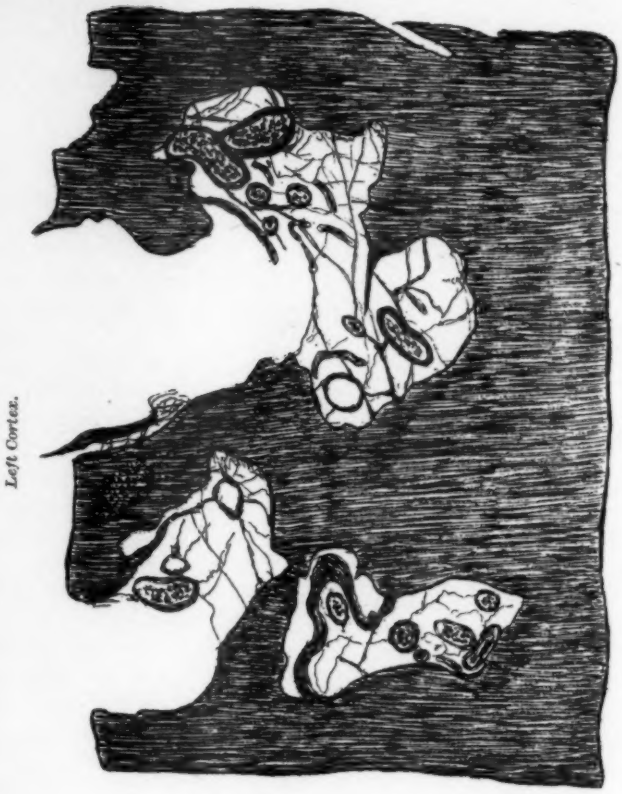
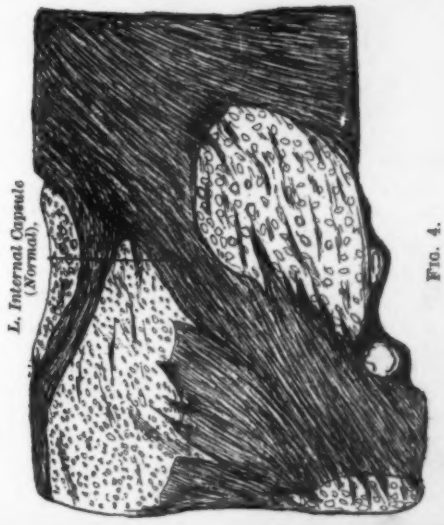


FIG. 3.

FIG. 5.



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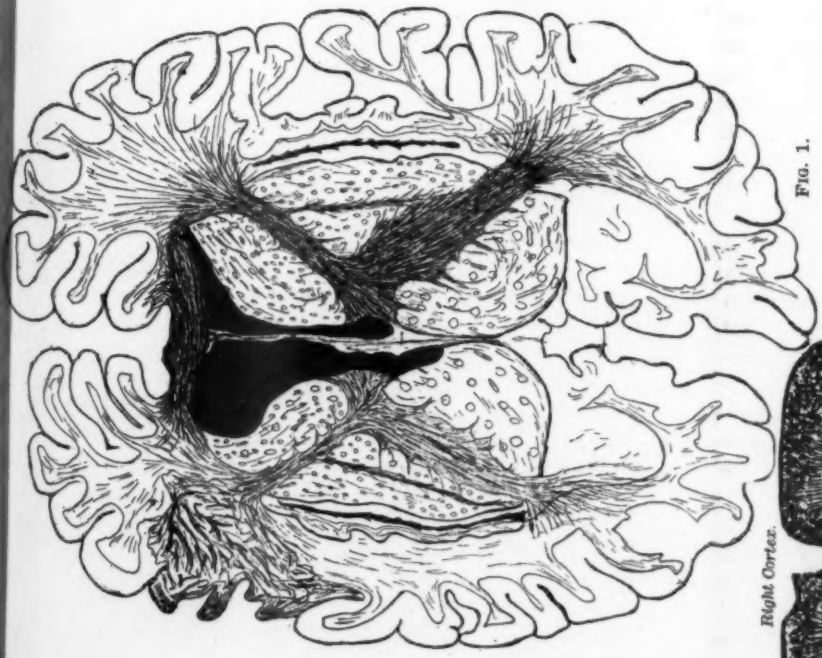


FIG. 1.

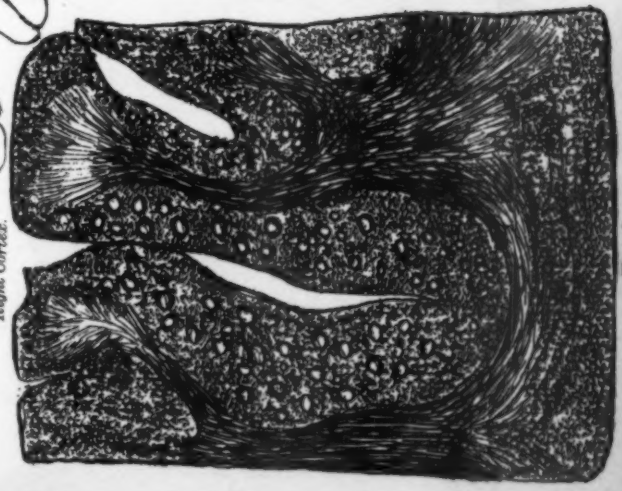
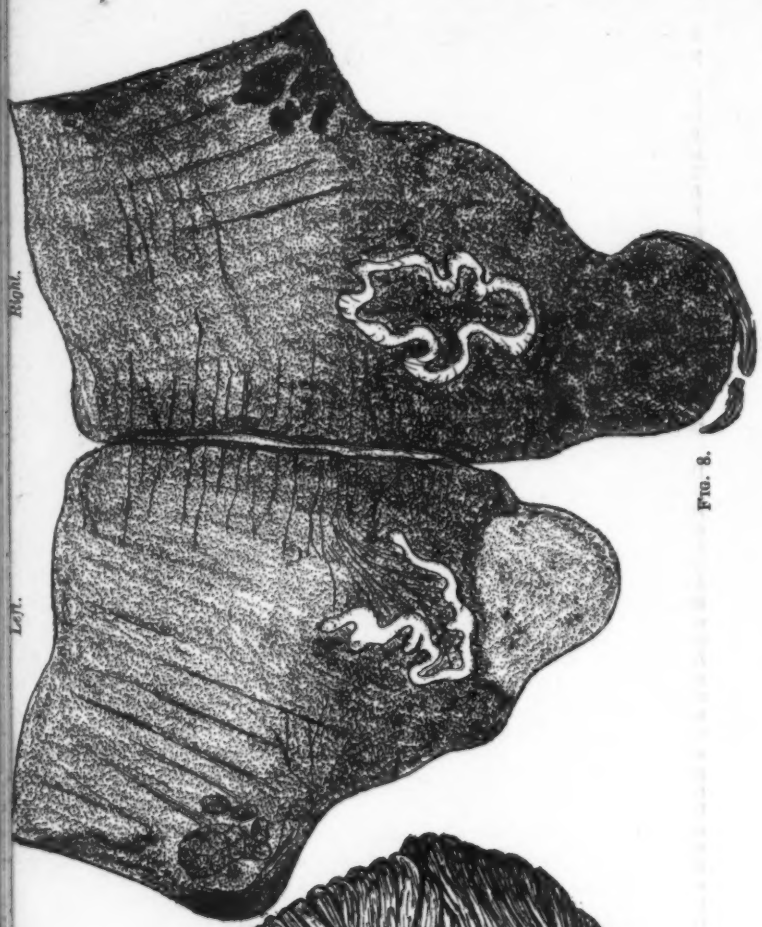


FIG. 2.



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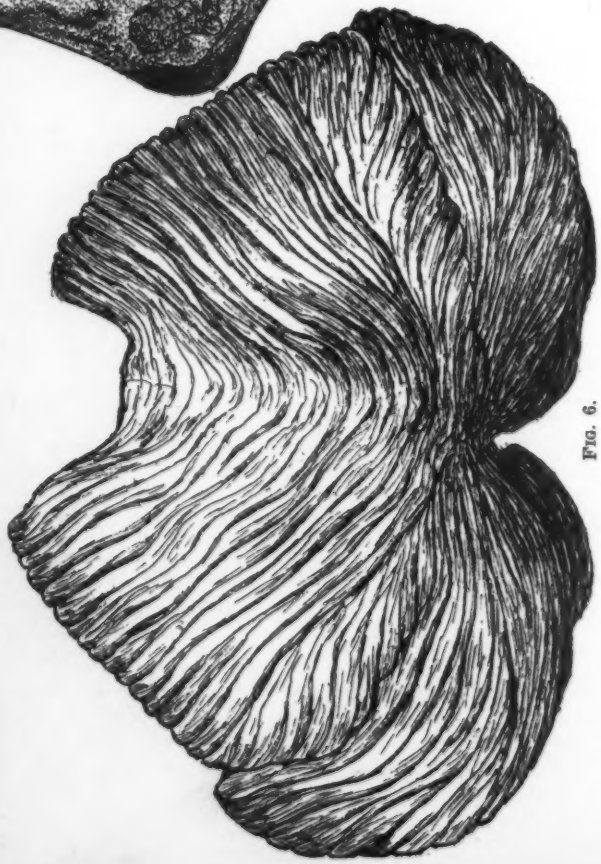

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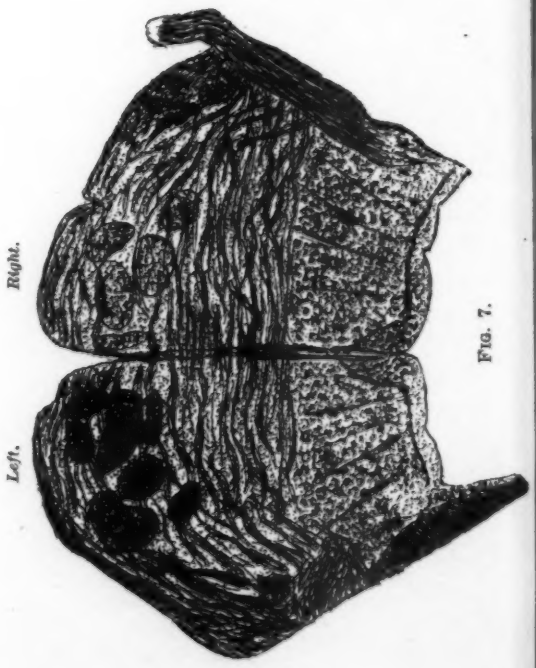

FIG. 7.

specimens as far down as the sacral region. The brain and sections of the spinal cord were placed in 10 per cent formalin.

The following is a description of the figures:

FIG. 1. This is a semi-diagrammatic drawing of a longitudinal section of the brain which passes through the lower second frontal region and includes the ventricles, the central nuclei as well as the internal and external capsules. For the more correct interpretation of the changes at the site of the lesions in the cortex and the changes in the capsular fibers and internal nuclei, sections stained by the Weigert-Pal method were used and the changes as seen under a dissecting microscope were added to the remaining entirely diagrammatic drawing.

One is immediately struck by the smaller dimensions and evident retraction of the entire left hemisphere. An almost total destruction of the tissue in the second frontal gyrus including the Island of Reil is found. A condition of a variable degree of microgyri is also evident in the cortex in the left frontal region. At the site of the lesion the blood vessels are increased and vessels of a larger size than are ever found in the normal gyri are found. The fibrous tissue infiltration is marked and its fibers rather coarse. The entire bundle of fibers making up the left internal capsule is about one-half the thickness of the right internal capsule. In the Weigert specimens by far the majority of the fibers of the left capsule stain only faintly, a few normal staining fibers being interspersed among them. The left lenticular caudate nuclei, especially the latter, are atrophied, also the left optic thalamus. The left lateral ventricle is moderately dilated.

FIG. 2. Weigert-Pal specimen of normal gyri taken from the right second frontal region, corresponding to the site of the lesion in the left frontal hemisphere.

FIG. 3. Weigert-Pal specimen with second Van Gieson staining. The section is from the site of the lesion in the second left frontal convolution. It shows a destruction of the greater portion of the gyrus; numerous cross-sections of blood vessels are seen, especially veins; coarse bands of connective tissue support these and replace to a certain extent the destroyed tissue. The formation of small cavities is seen, some lined by the dura, others by fibrous tissue. Considerable blood pigment is found on examining this specimen under the higher power and altogether there is no reasonable doubt but that a hemorrhage into the cortical substance was the cause of the pathological changes with formation of scar tissue and secondary atrophy of the pyramidal tract, internal nuclei and right hemisphere of the cerebellum.

FIG. 4. Weigert-Pal specimen from the region of the right internal capsule including portions of the optic thalamus, the lenticular and caudate nuclei. These appear normal and in marked contrast to the corresponding left region.

FIG. 5. Weigert-Pal specimen of the region of the left internal capsule including portions of the lenticular and caudate nuclei and optic thalamus. The atrophy of the internal capsule and optic thalamus is so evident in the drawing that further description is not needed. The oval and round clear spaces in the substance of the nuclei represent the enlarged perivascular spaces and lymph spaces about the nerve cells due to the retraction of the surrounding tissue.

FIG. 6. Represents the upper surface of the cerebellum. The well-marked atrophy of the right hemisphere is shown. This atrophy of the cerebellar hemisphere opposite to the side of the lesion has been described in several cases of cerebral infantile paralysis and it may be of interest to review briefly some of the opinions as to its cause. If we remember the existence of a fronto-cerebellar tract whose fibers pass from the motor region of one cerebral hemisphere to the cerebellar hemisphere of the opposite side, this change will be clear.

Gordinier gives the following description of this fronto-cerebellar tract. (Gordinier's Anatomy of the Central Nervous System, page 222.) "The fronto-cerebellar tract occupies rather more than the inner fifth of the crusta. The fibers of this tract come from the prefrontal lobe, and pass between the lenticular and caudate nuclei, occupying a large part of the anterior limb of the internal capsule, and a course downward on the inner side of the pyramidal tract, ending in the ventral portion of the pons Varolii; about the nerve cells of the nucleus pontis of each side are joined by fibers from the cortex of both cerebellar hemispheres, chiefly, however, with the cerebellar hemisphere of the opposite side. The fibers are the axones of the cells of Purkinje of the same and the opposite side, the latter fibers having crossed in the raphe, thus establishing a connection between the frontal lobe of the one side and both cerebellar hemispheres, but chiefly with the cerebellar hemisphere of the opposite side."

FIG. 7. Weigert-Pal section through the upper medulla showing the superficial and deep pontine fibers. As this section is above the decussation of the pyramidal tract, the degenerated fibers are found on the left side, in fact, the left half of the medulla is atrophied and even more so than represented in the drawing. The left pyramidal tract shows an advanced degeneration of its fibers so that few of them take up but a faint staining. The floor of the 4th ventricle is shallow and the nucleus of the hypoglossal nerves is seen close to the central raphe and just under the ventricular surface. In the deep pontine fibers of the left half of this section some atrophic changes are found.

FIG. 8. Weigert-Pal section through the medulla in the olivary region. In this section the atrophy of the left half of the medulla is still marked and more so than in figure 7. The left pyramidal tract is markedly atrophied and only a very few fibers stain at all. A few atrophic fibers are also seen in the lateral portion of the medulla above the large bundle of the pyramidal tract, these, no doubt, represent a subdivision of pyramidal bundles. The deep pontine fibers on the left side also show degenerative changes.

FIG. 9. Weigert-Pal section through the lower dorsal segment of the spinal cord. This shows a well marked degeneration of the right pyramidal tract as represented in the drawing by the lighter area in the postero-lateral portion of the cord. This area gradually shades into normal tissue and a narrow zone of normal fibers lies in the periphery of the cord, being fibers of ascending tracts. This degeneration in the crossed pyramidal tract on the right half of the cord can be traced to the lower sacral segment. Strange to say in none of the specimens from the medulla or spinal cord could a direct pyramidal tract be demonstrated and in reviewing the literature one finds that other observers have found it absent; no doubt, in such instances, all the motor fibers pass into the cord as a crossed tract. Sacki and Schmaus (Pathologie des Central Nervensystems, 1903, page 44), say: "We have previously mentioned the degenerative changes in the anterior direct pyramidal tract which lies on either side of the anterior sulcus of the cord. But this bundle does not at the present day hold the same importance it held in former years, in fact, it is at times impossible to demonstrate it in human beings." It is the best seen in lesions affecting the motor tract either in the internal capsule or crus cerebri or better still in those of the medulla or uppermost segments of the dorsal cord. Sacki and Schmaus also claim this tract has never been clearly demonstrated in lower animals. The following description of the anatomical relationships of the pyramidal tracts is taken from Gordinier's Anatomy of the Central Nervous System, page 91. "The motor fibers of the cord which are located in the direct and crossed pyramidal tracts arise from the motor areas of the brain, and represent the neuraxones of the large pyramidal cells, which are abundantly found in the third layer of the cortex. Their course from the cerebral cortex to the medulla will be described later. When they reach the medulla they occupy a large area on each side of the anterior median fissure, and at the first or second cervical nerves large bundles of fibers, or axones, representing about eighty per cent of the whole number, pass obliquely across to the opposite side, entering the posterior part of the lateral column of the cord, hence the name 'crossed pyramidal tract.' These crossed fibers become vertical and extend downward, gradually decreasing in size until they reach their termination at the level of the third or fourth sacral nerve, a small number of fibers continuing downward to terminate in the filum terminale. The neuraxones which do not cross representing about twenty per cent of the motor fibers, pass downward in the area of the cord adjacent to the anterior median fissure on the same side; hence they are called the direct or uncrossed pyramidal tract. They usually cease about the level of the mid-dorsal region. The motor neuraxones, like most of the long fibers of the columns of the cord, give off at different levels side branches or collaterals, which leave the parent stem at right angles. The axones, with the collaterals composing the crossed pyramidal tract of each side, pass forward and inward, entering the gray matter, where they break up about the motor nerve cells into innumerable fine filaments or aborizations. The neuraxones and collaterals of the direct pyramidal tract end, according to Lenhossek, in fine brush-like expansions about the motor nerve cells of the anterior horn of the same side. On the contrary, undoubted clinical and experimental evidence is at hand to prove that the greater portions of the fibers cross over through the anterior commissure to end about the motor nerve cells existing in the opposite anterior cornu. Most of the fibers of the direct pyramidal tract seem destined to the arm; hence the relation of the arm is almost exclusively with the opposite side. The fibers

of the tracts, direct and crossed, conduct impulses of voluntary motion from the motor areas of the brain to the muscles. If the fibers of the motor tract be destroyed by severing their connection with the cells of the motor area of the brain, there will result a motor paralysis of the opposite side of the body and a descending degeneration from the point of lesion throughout the entire extent of the tract. In the cord the degenerated areas will be the direct pyramidal tract of the same, and the crossed pyramidal tract of the opposite side. This degeneration is complete, involving the termination of the axones and collaterals, about the nerve cells of the anterior cornua, and is due to the loss of trophic or nutritional influence, which results from the severance of the nerve fibers from their mother cells, in the motor areas of the cortex. The peripheral portions of the tract on the contrary remain normal, because its nutrition is dependent upon the motor cells of the anterior cornu whose neuraxones form the peripheral portions of the tract." For a more complete discussion of the subject of infantile cerebral palsies, especially their relationship to the acute exanthemata and infectious diseases in general, the reader is referred to the article of Freud (Nothnagel's Specielle Pathologie und Therapie, Vol. IX, Part 3); that of Osler (Medical News, 1888, No. 2, 3, 4 and 5); Strümpell, Ueber die Acute Encephalitis der Kinder (Jahrbuch für Kinderheil Kunde, XXII, 1884); Strümpell on primary acute encephalitis (Deutsches Archiv für klin. Medicin, Vol. XLVII); also P. Marie, hemiplegie cérébrale infantile et Maladie infectieuses (Progrès méd., 5 Sept., 1885).

NOTES AND NEWS.

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NOTES ON NEW BOOKS.

New Methods of Treatment. By DR. J. LAUMONIER. Translated and edited from the second revised and enlarged French edition by H. W. STERS, M. A., M. D. (*Chicago: W. T. Keener & Co., 1904.*)

This work treats in a general way of the recently introduced remedies. One cannot help wondering, on going over the book, whether the consideration of many of these is worthy of being kept in a permanent form. Of the large number of newer remedies described only a very few will be used by more conservative therapeutists. Considerable space is given to the discussion of various glandular extracts, but regarding these there is nothing specially new brought forward. The same may be said about the use of the various serums. One does not find anything of marked interest in this section, and under the heading "anti-typhoid inoculation," there is no mention whatever of the work of Wright. Notes regarding some of the newer drugs may be found useful, but one cannot help feeling that all that is valuable in this book might have been put in a quarter of the space. It is written in an agreeable style and the translation has been well done.

Medical Diagnosis. JOHN H. MUSSER, M. D. 5th edition. (*Lea Bros. & Co., 1904.*)

The early appearance of the fifth edition of Dr. Musser's book gives evidence of its extensive use in the profession. The present edition contains over one hundred more pages and one hundred and fifty-nine more illustrations than the last, making the book reach the upper limit of size practicable for a single volume.

The quality of the illustrations is variable, the wood-cuts being generally good, as are some of the colored plates, those picturing gross specimens, sputum, culture tubes, etc., being rather below the mark of excellence.

The general arrangement of the text has been maintained although some minor changes appear. Some chapters have been considerably expanded.

Among these, that treating of Internal Parasites has been rewritten and enlarged by the addition of some good cuts of parasites' eggs, etc. The subject is well brought up to date by the consideration of the newer parasitic forms such as *Uncinaria americana* and those of the trematode worms which have become of interest through our closer relations with the Far East.

The press work is good and the volume will prove acceptable to many students and practitioners who desire a general reference work on all phases of clinical diagnosis.

Appleton's Medical Dictionary. An illustrated dictionary of medicine and allied subjects in which are given the derivation, accentuation and definition of terms used throughout the entire field of medical science. Edited by FRANK P. FOSTER, M. D. (*New York and London: D. Appleton & Co., 1904.*)

If the present activity in the making of medical dictionaries continues, there seems little danger that the supply will not equal the demand. The volume before us of nearly 2000 pages seems more of a dictionary and less of an illustrated cyclopedia than almost any other upon the market. It represents conservatism in lexicography, and is prepared on strict lexicological lines. In the printing of words full weight is given to diphthongs; as, for example, œdema, œsophagus, hæmorrhage, and the like. The number of medical words given is very large and the book in fact, is somewhat cumbered with words, many obsolete terms being given. There is also a tendency to use unaccustomed words, often constructed on unimpeachable etymology in place of those which have become generally accepted; as, for example, *ecphyaditis* instead of *appendicitis*. The definitions are excellent and can be heartily praised. Certain definitions, however, are omitted; for instance, *dementia præcox* in the sense used by Kraepelin is not given, the nearest approach being the *acute dementia* of Pinel. The terms used by Ehrlich for example, to describe his side-chain theory of immunity, are many of them lacking, and one is compelled to conduct an elusive search through the dictionary and the supplement without getting after all any very clear idea as to their real meaning. Some of the newer terms of physiological chemistry are also lacking. The definitions of botanical terms are full and satisfactory.

The illustrations are not of a high order of merit, and the majority of them could have been omitted. The book itself is well and clearly printed. The volume as a whole is to be highly commended and its scope and execution remind one of the two first volumes of Foster's Encyclopedic Medical Dictionary which will always remain a monument to the learning and thoroughness of the editor.

Lectures to General Practitioners on the Diseases of the Stomach and Intestines. By BOARDMAN REED, M. D. Illustrated. (*New York: E. B. Treat & Co.*)

In his preface the author states that the majority of physicians now in practice have had no opportunity of learning the newer methods available in the diagnosis and treatment of diseases of the digestive system, and that books describing them are necessary. From this one might conclude that such books possibly do not exist. The writer, however, announces himself that books upon this subject have been multiplying during the last ten years, and that the subject has been treated most ably and exhaustively by several American writers, to say nothing of the works by foreign authors, which have been translated into English. Under the circumstances, it would therefore seem that the present work might have been dispensed with; but again the writer explains that there does not at present exist in the English language any work which in a single volume treats of the whole subject of diseases of the stomach and intestines from the standpoint of our present knowledge, and that the general practitioners and specialists in other lines desire such a work in a single volume. This, therefore, is the *raison d'être* of the author's work. It is written in the form of lectures, which are based in part upon those delivered to classes in the department of medicine of Temple College, Philadelphia, and in part upon his "Talks to General Practitioners" contributed by the writer to the International Medical Magazine, during the five years that the journal was under his editorial management.

Bearing in mind the standpoint from which the book is written, it may be considered a success, for it contains all those data, which the general practitioner is apt to look for in a work of this kind. It contains much therapeutic information, and considers besides questions of diet such resources as electrostatic currents and X-rays, the violet rays, the use of radium, mechanical vibration, manual therapy, hydrotherapy, exercise, etc.

In the accompanying prospectus of the publishers it is pointed out that in the writer's intercourse with chronic dyspeptic and nervous invalids, many of whom were exceedingly irritable and intolerant of all but the gentlest handling, the writer was led to devise methods as little unpleasant and disturbing as possible, compatible with accurate diagnosis and successful treatment. Such methods are to be found in the work before us.

A novel as well as useful feature is the introduction of a symptomatic guide to diagnosis, so far as the symptoms have to do with the gastro-intestinal apparatus.

As we have already suggested, the book will prove useful to the general practitioner for whom it is intended, but it is not a scientific work and often somewhat disappointing from this standpoint. Objectionable, and not in good taste is the ubiquitous and insistent appearance of the writer's Ego.

The publishers' work has been well done, so far as the text goes; the illustrations, however, which for the most part have been borrowed, are rather poor.

Essentials of Nervous Diseases and Insanity: their Symptoms and Treatment. By JOHN C. SHAW, M. D., late Clinical Professor of Diseases of the Mind and Nervous System, Long Island College Hospital Medical School. Fourth Edition. Thoroughly Revised. By SMITH ELY JELLIFFE, Ph. G., M. D., Clinical Assistant, Columbia University, Department of Neurology; Visiting Neurologist, City Hospital, New York, 12mo. Volume of 196 pages, fully illustrated. (*Philadelphia, New York, London: W. B. Saunders & Company, 1904.*)

This is a very creditable book which must be of great service to the medical student. The portion relating to the diagnosis of insanity is especially to be commended as it gives the student the latest views of an ever-shifting subject.

Essentials of Anatomy; including the Anatomy of the Viscera. By CHARLES B. NANCREDE, M. D., Professor of Surgery and Clinical Surgery in the University of Michigan, Ann Arbor. Seventh Edition. Thoroughly Revised. (*Philadelphia, New York, London: W. B. Saunders & Company, 1904.*)

In commenting on a previous edition we have had occasion to speak favorably of this students' manual. The present edition has been carefully revised and the anatomy of the nervous system has been especially considered. The book is designed expressly for students who are called upon to review their anatomy.

A Compend of Medical Latin Designed Expressly for Elementary Training of Medical Students. By M. T. ST. CLAIR, A. M., Professor of the Latin Language, etc. Second edition, revised. (*Philadelphia: P. Blakiston's Son & Co., 1904.*)

This is a clearly written and practical little compend of medical Latin prepared expressly for those who have entered upon the study of medicine without a previous knowledge of the language. At best the knowledge which can be acquired even from the best compend of this character is a make-shift and not in the line of thoroughness. If a student must secure a modicum of Latin and is satisfied with it, we know of no short treatise which will prove as useful as the one under review. The list of abbreviations is full and the vocabulary is good.

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State Board of Health of the State of Ohio. Seventeenth Annual Report. For the year ending December 31, 1902. 8vo. 508 pages. 1904. Springfield, Ohio.

A Text-Book of Human Physiology. By Albert P. Brubaker, A. M., M. D. With colored plates and 354 illustrations. 1904. 8vo. 699 pages. P. Blakiston's Son & Co., Philadelphia.

Appleton's Medical Dictionary. An Illustrated Dictionary of Medicine and Allied Subjects, in which are given the Derivation, Accentuation and Definition of Terms Used Throughout the Entire Field of Medical Science. Edited by Frank P. Foster, M. D. 1904. 4to. 1991 pages. D. Appleton and Company, New York and London.

A System of Practical Surgery. By Prof. E. von Bergmann, M. D., Prof. P. von Bruns, M. D., and Prof. J. von Mikulicz, M. D. Volume IV. Translated and edited by William T. Bull, M. D., Edward Milton Foote, M. D., Carleton P. Flint, M. D., and Walton Martin, M. D. *Surgery of the Alimentary Tract.* 1904. 8vo. 755 pages. Lea Brothers & Co., New York and Philadelphia.

The Principles and Practice of Gynecology. For Students and Practitioners. By E. C. Dudley, A. M., M. D. Fourth edition, revised. With 419 illustrations in colors and monochrome, of which 18 are full-page plates. 1904. 8vo. 771 pages. Lea Brothers & Co., Philadelphia and New York.

Saunders' Question-Compends, No. 21. Essentials of Nervous Diseases and Insanity. Their Symptoms and Treatment. By John C. Shaw, M.D. Fourth edition thoroughly revised by Smith Ely Jelliffe, M. D., Ph. D. Illustrated. 1904. 12mo. 196 pages. W. B. Saunders & Co., Philadelphia, New York, London.

Saunders' Question-Compends, No. 7. Essentials of Materia Medica, Therapeutics and Prescription Writing. Arranged in the form of questions and answers. Prepared especially for students of medicine. By Henry Morris, M. D. Sixth edition, thoroughly revised by W. A. Bastedo, Ph. G., M. D. 1904. 12mo. 294 pages. W. B. Saunders & Co., Philadelphia, New York, London.

Saunders' Question Compends, No. 3. Essentials of Anatomy. Including the Anatomy of the Viscera. Arranged in the form of questions and answers. Prepared especially for students of medicine. By Charles B. Nancrede, M. D. Seventh edition, thoroughly revised. 1904. 12mo. 419 pages. W. B. Saunders & Co., Philadelphia, New York, London.

Clinical Lectures on Mental Diseases. By T. S. Clouston, M. D., Edin., F. R. C. P. E. Sixth edition. 1904. 12mo. 738 pages. Lea Brothers & Co., Philadelphia and New York.

Physician versus Bacteriologist. By Prof. Dr. O. Rosenbach, of Berlin. Authorized translation from the German by Dr. Achilles Rose. 1904. 12mo. 462 pages. Funk and Wagnalls Company, New York and London.

Hand-Book of the Anatomy and Diseases of the Eye and Ear. By D. B. St. John Roosa, M. D., LL. D., and A. Edward Davis, A. M., M. D. 1904. 16mo.. 297 pages. F. A. Davis Company, Philadelphia.

Arteria Uterina Ovarica. The Utero-Ovarian Artery, or The Genital Vascular Circle. Anatomy and Physiology, with their Application in Diagnosis and Surgical Intervention. By Byron Robinson, B. S., M. D. 1903. 4to. 182 pages. E. H. Colegrove, Chicago, Illinois.

Refraction and How to Refract. Including Sections on Optics, Retinoscopy, the Fitting of Spectacles and Eye-Glasses, etc. Third edition. Two hundred and fifteen illustrations, thirteen of which are colored. 1904. 8vo. 314 pages. P. Blakiston's Son & Co., Philadelphia.

A System of Practical Surgery. By Prof. E. von Bergmann, M. D., Prof. P. von Bruns, M. D., and Prof. J. von Mikulicz, M. D. *Volume V. Surgery of the Pelvis and the Genito-Urinary Organs.* Translated and edited by William T. Bull, M. D., and Edward Milton Foote, M. D. 1904. 8vo. 789 pages. Lea Brothers & Co., New York and Philadelphia.

Practical Dietetics. With Reference to Diet in Disease. By Alida Frances Pattee. Second edition, revised and enlarged. 1904. 12mo. 311 pages. Published by the Author, New York City.

A Treatise on Obstetrics. For Students and Practitioners. By Edward P. Davis, A. M., M. D. Second edition. Illustrated with 274 engravings and 39 plates in colors and monochrome. 1904. 8vo. 809 pages. Lea Brothers & Co., Philadelphia and New York.

A Practical Treatise on Diseases of the Skin. For the Use of Students and Practitioners. By James Nevins Hyde, A. M., M. D., and Frank Hugh Montgomery, M. D. Seventh and revised edition. Illustrated with 107 engravings and 34 plates in colors and monochrome. 1904. 8vo. 938 pages. Lea Brothers & Co., Philadelphia and New York.

Diseases of the Nose, Throat and Ear, and their Accessory Cavities. By Seth Scott Bishop, M. D., D. C. L., LL. D. Third edition, thoroughly revised and enlarged. Illustrated with ninety-four colored lithographs and two hundred and thirty additional illustrations. 1904. 8vo. 564 pages. F. A. Davis Company, Philadelphia.

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International Clinics. A Quarterly of Illustrated Clinical Lectures and Especially Prepared Original Articles on Treatment, Medicine, Surgery, Neurology, Pediatrics, Obstetrics, Gynecology, etc. Edited by A. O. J. Kelly, A. M., M. D. Volume III. Fourteenth Series, 1904. 8vo. 302 pages. J. B. Lippincott Company, Philadelphia.

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A Compend of Medical Latin. Designed Expressly for Elementary Training of Medical Students. By W. T. St. Clair, A. M. Second edition, revised. 1904. 12mo. 131 pages. P. Blakiston's Son & Co., Philadelphia.

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Multiple Personality. An Experimental Investigation into the Nature of Human Individuality. By Boris Sidis, M. A., Ph. D. (Harvard), and Simon P. Goodhart, Ph. B. (Yale), M. D. 1905. 8vo. 462 pages. D. Appleton and Company, New York.

Medical Laboratory Methods and Tests. By Herbert French. 1904. 16mo. 152 pages. W. T. Keener & Co., Chicago.

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